HUMAN
INTESTINAL PROTOZOA
IN THE NEAR EAST

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WELLCOME
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AN INQUIRY INTO SOME PROBLEMS AFFECTING THE SPREAD AND INCIDENCE OF INTESTINAL PROTOZOAL INFECTIONS OF BRITISH TROOPS AND NATIVES IN THE NEAR EAST, WITH SPECIAL REFERENCE TO THE CARRIER QUESTION, DIAGNOSIS AND TREATMENT OF AMŒBIC DYSENTERY, AND AN ACCOUNT OF THREE NEW HUMAN INTESTINAL PROTOZOA

[*Conducted under the auspices of the Medical Advisory Committee, M.E.F. (January to August, 1916)*]

BY

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PREFATORY NOTE.

THE "Report on the Human Intestinal Protozoa in Egypt" was completed during the months of July, August and September, 1916, and was sent to the War Office in October. Parts I.–IV. were published in the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS (Nos. 1—6, January to June, 1917). Lack of space did not permit the publication of Part V., which appears here as the Appendix for the first time. Thanks to Mr. H. S. Wellcome, who has already done so much to help the investigation of dysentery problems in England during the War, it has been possible to publish the complete Report with a few additions and corrections, together with a paper on "The Carriage of Cysts of *Entamœba histolytica* and other Intestinal Protozoa by House-flies," which was first issued in Egypt in April, 1916, by order of the Director of Medical Services as a memorandum for circulation amongst medical officers. It was republished in the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS in May, 1917. The section of the Report in Part IV., which deals with the experiments on house-flies as carriers of cysts of protozoa, has reference to this memorandum which had appeared previously.

Colonel W. H. Horrocks, C.B., K.H.S., Editor of the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS, has very kindly permitted the re-printing of those sections of the Report and the paper on the fly carriage of cysts which have already appeared as explained above.

C. M. W.

1917. F. W. O'C.

INTRODUCTION.

The work carried out in Egypt, of which this paper forms the report, was undertaken at the request of Surgeon-General Sir William Babtie, V.C., K.C.M.G., at that time P.D.M.S., M.E.F., in consultation with the Medical Advisory Committee. Its object was threefold, namely, an investigation into the carrier problem of amœbic dysentery amongst the troops with a view to the possible elimination of the carriers; secondly, an inquiry into the best method of administering emetin to carriers and actual dysenterics with the object of establishing some uniform line of treatment which would give the maximum of good result; and, thirdly, an examination of the fly transmission of amœbic dysentery by means of cyst carriage, and a determination of the best means of destroying the resistant amœbic cysts after their escape from the body.

During the inquiry into the three problems named above, although our attention was chiefly directed towards the pathogenic *Entamœba histolytica*, all the various intestinal protozoa of man in Egypt came under observation, and we have been able to gather a large amount of information regarding them. Much of this is quite new, and it will be set forth in the following pages under different headings.

The work was carried out at Alexandria, as Surgeon-General Babtie rightly considered this to be the most suitable locality for an investigation of this kind amongst troops in camp. We were fortunate at the outset in obtaining a very excellent laboratory at the Orwa-el-Waska Section of the 19th General Hospital, where Lieutenant-Colonel Scott, R.A.M.C., officer commanding in charge, and Captain Lambkin, R.A.M.C., Registrar of 19th General Hospital and officer commanding at Orwa-el-Waska, gave us every assistance. It is not too much to say that without the help of these officers and their readiness on every occasion to carry out our wishes, the work we had in hand could not have been so successfully conducted. For the treatment of the cases we had two

dysentery wards at our disposal, and we were able to observe closely the course of the various infections and the effect of treatment. The late Lieutenant-Colonel Lister, C.M.G., R.A.M.C., Medical Superintendent of the Hospital, who always showed a lively interest in our investigations, very kindly allowed us to take over the treatment of all the amœbic or other protozoal infections, so we had ample material on which to work.

In addition to the cases which were identified by us in the routine examination of all the hospital admissions, we obtained a larger number of cases from the examinations of apparently healthy men in various camps around Alexandria. As will be shown below, we found a fairly high percentage of carriers amongst the healthy or apparently healthy men, and these were brought into hospital and given a course of emetin, the result of which will be discussed in another part of this paper.

The carrying out of the routine examinations of men in camp seemed at first sight a very difficult procedure, but a system was elaborated which proved to be reliable chiefly owing to the interest shown by Captain Sibley, R.A.M.C., then Sanitary Officer to one of the camp areas, who helped us in every possible way and rendered easy what appeared at first sight an almost impossible undertaking. The working of any scheme for the collection of material from men in camp is no easy matter. Yet the energy and skill of Serjt. E. Weavis, R.A.M.C., specially detailed for laboratory duty, enabled us to collect several thousand specimens without any mistake or hitch in the proceedings.

In order to obtain an insight into the intestinal protozoa of the natives of the country, with a view to the discovery of a reservoir of infection, we were kindly given permission by Dr. Kirton, Medical Superintendent of the Egyptian Jails, to examine a series of over 500 prisoners in the Hadra Prison, Alexandria. These observations afforded most interesting results which will be detailed below.

The patients in hospital were under the care of a medical officer who carried out the treatment suggested by us. For the greater part of the time Lieutenant Eastmond, R.A.M.C., was in

charge, and our thanks are due to him for the trouble he took with our cases. As will be explained below, they were controlled very carefully, at least one entire stool from each being inspected daily. We obtained detailed histories from all the cases, especially as regards the possibility of past dysentery or emetin treatment, and the occurrence of any symptoms during the observation was carefully noted. In the laboratory work connected with these hospital cases, which involved blood examinations, bacteriological culture work, collection of material and obtaining information from the patients, we have been very much helped by Cpl. W. J. Muggleton, R.A.M.C., who was senior laboratory assistant at the Wellcome Bureau of Scientific Research before he left for active service abroad, in connection with our work.

When the hospital cases had completed their course of treatment it was necessary to keep them under observation for some time. An arrangement was made whereby a section of the convalescent depot at Mustapha was set apart for their reception. From these cases Serjt. Weavis collected specimens on alternate days for at least one month, so that we were able to note at once when any relapse of the infection occurred. Major Fisher, R.A.M.C., Officer Commanding the Convalescent Depot at Mustapha, made all arrangements for the reception of our cases, and we are greatly indebted to him for the trouble he took and the care with which our wishes were carried out.

We wish also to express our indebtedness to Colonel Beach, C.M.G., A.D.M.S., at Alexandria, for the interest he took in our inquiry and for the help and assistance he constantly gave, especially in smoothing our path in the carrying out of what were sometimes rather difficult and intricate negotiations. Finally, we wish to acknowledge the help we received from Colonel Sandwith, C.M.G., whose wide Egyptian experience and the position he held as Consultant Physician to the Military Hospitals in Alexandria, rendered his assistance and encouragement most valuable.

PART I.*

The Incidence of Protozoal Infections amongst British Troops and Natives in Egypt with Special Reference to the Carrier Problem of Amœbic Dysentery.

The examinations for intestinal protozoa were carried out upon several classes of men, including healthy British troops on full duty in camps round Alexandria, healthy British and British West Indian cooks employed in the same localities, British convalescents in the convalescent camp at Mustapha, healthy native prisoners in the Hadra Prison, British prisoners in Gabarri Prison, the permanent Royal Army Medical Corps staff of the Convalescent Depot at Mustapha, the Royal Army Medical Corps staff of the Orwa-el-Waska Section of the 19th General Hospital, and all the cases admitted to the Orwa-el-Waska Hospital for dysentery and other intestinal disorders, the majority of the latter coming from various stations in Egypt, but some being invalids from Mesopotamia. The various findings in these several groups will be discussed below, but first of all we will describe the methods of our examinations and the value of these in determining a correct percentage of the various infections.

Method of collecting Material.

In the case of hospital patients there is no difficulty in obtaining samples of the stool for examination. We arranged that the entire stool was brought for inspection in a bed-pan, a much more satisfactory procedure than when only a small sample is sent from the ward in a tube. An inspection of the entire stool gives a much more accurate idea of the condition of the patient. When only small samples are sent small quantities of blood and mucus are liable to be overlooked and one fails to get a correct notion of the character of the entire stool. In order to do this it is essential to have some room or lavatory near the laboratory to which pans can be brought for inspection.

When it comes to the collection of material from men in camps or prisons much greater difficulties have to be encountered. These can only be surmounted by interesting the men in the proceedings and it is essential to have some reliable person in authority to carry

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* Reprinted from the *Journal of the Royal Army Medical Corps*, January, 1917.

out the arrangements and to control the men. We were fortunate in having the services of Serjt. Weavis, who carried out his duties admirably. Arrangements were made through the sanitary officers of the camps or prisons who saw that a special latrine or screened area was set apart for the purposes of collection. Within the enclosure were arranged in a row a series of "stool closets," which are small zinc pans each supported on an iron ring on four metal legs. There is a wooden seat round the pan. We found that a couple of dozen of these were ample for working purposes. The men from whom samples are to be collected are paraded, preferably directly after breakfast, near the enclosure. They are then instructed, numbered off and told to use the stool closet corresponding to their numbers. The men are ordered not to micturate into the pan, into which only fæces are to be passed. Each man is given a collecting tube with a metal spoon attached to the cork or some other receptacle, and when he has eased himself he takes a sample of the stool on the metal spoon and places it in the tube. During the proceedings it is necessary to have an orderly, who understands the work, on duty in the latrine to see that the men carry out the process properly and do not attempt to mix up the samples either by accident or intentionally. The men carrying the tubes line up outside and the name of each is written on the tube while the serjeant obtains any information such as history of dysentery, previous foreign service and so on. Meanwhile a number of cleaners are occupied with the zinc pans and when they are properly cleaned and dried they are replaced and another series of men instructed to repeat the procedure. In this manner in a very short time it is possible to collect reliably fifty or sixty specimens. It is not possible, of course, to collect a sample from every man paraded, for a certain number of men will be unable to oblige. The "defaulters" can be again paraded with a fresh lot of men the following morning. This method of collection we have used regularly and it has yielded very good results. The men who were found to be carriers of *E. histolytica* were ordered into hospital for treatment, and, though this was done on the evidence of a single sample collected in the manner described above, in no case did an error arise in the matter of bringing the wrong man into hospital.

We have described the method in some detail, because, when it was at first suggested that we should collect samples from healthy men it was thought that the difficulties to be overcome would be insurmountable. Furthermore, the method could be employed for

the collection of samples for many other purposes, as, for instance, examinations of typhoid carriers.

In all the examinations of men in camp we have examined only single samples from each man. We recognize that in so doing a number of cases of infection are missed, for the protozoal infections of the human intestine are very irregular as judged by the appearance of the protozoa in the stool.

The Value of a Single Examination in determining Infections.

If one looks through the protocols of the cases of *E. histolytica* infection at the end of this report it will at once be seen that many protozoal infections which were not apparent at the first examination of a patient's stool appeared later on. This was true of all the protozoa found in the intestine. On several occasions cases which have been treated for a lamblia or other infection have, during the control, suddenly given evidence of an infection of *E. histolytica*. Such observations prove to us clearly that a single examination of any individual may give an erroneous view of the infection. In order to arrive at some idea of the error we undertook the examination of a group of healthy men. The permanent Royal Army Medical Corps staff of the Mustapha Training Depot were chosen for this purpose. A series of ninety-two men were examined for a number of days. It was intended originally to examine them all every day, but owing to the changes in the staff and the movements of troops this was impossible. However, some of the men were examined twelve times, others eleven, others ten, and so on during the twelve days, and the results have been arranged in Table I, in the order of the number of examinations. In the first column have been placed the complete findings resulting from all the examinations and in the twelve columns following the result of the daily examinations. Thus case Thomas was found to have *E. coli* cysts (E.c.c.), *E. histolytica* cysts (E.h.c.), free amœbæ (E.f.), lamblia cysts (L.c.), *E. nana* cysts (E.n.c.), and *E. nana* free (E.n.f.). On the first examination, however, were found only *E. coli* cysts, lamblia cysts and *E. nana* cysts, and it was not till the sixth examination that *E. histolytica* cysts appeared. It will be noted that the majority of cases examined yielded results of this kind. Thus, looking at the *E. histolytica* infections alone, it will be seen that this parasite was found in twelve of the ninety-two cases, but that in only four of these was it found at the first examination. If we are to accept this finding it would mean that all the figures resulting from a

single examination would have to be multiplied by three in order to arrive at a correct result. Whether this is too high an estimate future investigations will show, but it seems to us that the findings of a single examination are far below what they really should be and that to triple the figures would give a result much more nearly accurate. The findings amongst the various groups to be described now are all based on the single examination, with the possible exception of a very small number of hospital cases which have been examined two, three, or even more times. It will be realized that the figures, though they may appear high, are very much lower than they would have been if the examinations had been repeated.

One important point requires mentioning, and that is the possibility of the errors of a single examination being less when the organism is the actual cause of disease. For instance, when lamblia is the cause of diarrhœa with mucus, it is present in enormous numbers and there is no chance of missing it on first examinations. It is only when it is not producing symptoms that it is likely to be overlooked. Similarly, if *E. histolytica* is giving trouble, the probability is that the amœbæ or its cysts will be found at once. These errors, therefore, in the single examinations do not appear, or appear to a much smaller extent in those cases where the organism looked for is the actual cause of trouble. The error is the greatest in the search for carriers, and it is a consolation to know that if any infection is missed it is unlikely to have been the cause of trouble to the individual himself at the particular time, though it must never be forgotten that the *E. histolytica* carrier is the constant source of infection for others.

It should be mentioned that the records in Table I are based on thorough and careful examinations. For example, the time spent on the daily examination of any one case was not less than ten minutes, and in most instances at least two or three films were subjected to a close scrutiny.

Examination of Various Groups of Men for Protozoal Infections.

(*a*) *Healthy Troops.*—An examination of a large number (1,979) of healthy men on full duty was undertaken with the object of discovering the percentage of carriers of various protozoa, especially of *E. histolytica*. The samples for examination were collected in the manner described above. The following groups were examined :—

Metras camp, 1,013 men.

Mustapha Convalescent Depot, permanent staff, 312 men.

TABLE I.—A Series of Cases to illustrate the Error involved in a Single Examination—Infections which are not apparent at the First Examination appear later. The Complete Finding appears in Column A, the Daily Findings in Columns 1 to 12.

	A	1	2	3	4	5	6	7	8	9	10	11	12
Pavis	E.c.c.	−	−	−	−	+	+	−	+	−	−	−	−
	E.f.	−	+	−	+	−	+++	−	−	−	+	−	−
Howard	E.c.c.	+	..	+	+	−	−	−	−	−	+++	+	−
	E.f.	−	..	−	−	+	−	+	−	−	−	−	+
Beaumont ..	E.c.c.	−	++	−	+++	+	−	..	−	+	++	−	+
	E.f.	−	−	−	−	−	−	..	+	+	−	−	−
	Tet. c.	−	−	++	−	−	−	..	−	−	−	−	−
	Tet. f.	−	−	++	−	−	−	..	−	−	−	−	−
Bamford	E.n.c.	−	−	−	−	−	−	−	..	−	+	−	+
Thomas	E.c.c.	+++	++	+	−	++	−	−	−	+	−	..	+
	E.h.c.	−	−	−	−	−	++	−	+	+	+	..	−
	E.f.	−	−	−	+	−	+	+	−	−	−	..	−
	L.c.	++	+	+	−	+	+	+	++	+	+	..	+
	E.n.c.	+++	−	+	+	++	++	−	++	+	+++	..	+++
	E.n.f.	−	+++	−	+	−	−	−	−	−	−	..	−
Reenie	−	..	−	−	−	−	−	−	−	−	−	−	−
Licrave	E.c.c.	++	−	+	+	−	−	−	−	−	++	+	−
	E.f.	−	−	−	−	+	−	+	−	−	−	+	−
Wellwood	E.c.c.	+	++	++	++	+	++	..	+	..	+	+	+
Hewlett	E.c.c.	+	++	+	..	−	+	+	+	−	..	−	−
	E.f.	−	−	−	..	−	+	−	−	+	..	+	+++
	E.n.c.	−	−	−	..	−	−	+	−	−	..	−	−
Price	Trich.	−	..	−	+	−	−	−	−	−	−	..	−
Ferrie	Tet.c.	+++	+++	+	−	−	−	−	..	−	−	++	..
	Tet.f.	−	+++	+++	+++	+++	++	+++	..	+++	+++	−	..
Bailey	E.c.c.	−	−	−	..	−	−	−	−	..	−	−	+
	E.f.	−	−	+	..	+	−	−	+	..	−	−	−
Baker	E.c.c.	..	+++	..	+	+	+	+	+	−	+	+	++
	E.f.	..	−	..	−	−	−	−	−	+	++	−	−
	E.n.c.	..	−	..	−	−	−	−	+	−	−	−	−
Fursk	E.f.	..	+	+	+	−	+	+	−	..	−	−	++
	E.n.c.	..	−	−	−	−	−	−	−	..	++	+++	+++
	E.n.f.	..	−	−	−	−	−	−	−	..	+++	+++	+++
	Tet.c.	..	−	+++	−	−	+	−	−	..	+	−	−
	Tet.f.	..	+++	+	+++	+++	++	+	+	..	+	+++	−

Hare	L.c.	..	+++	+++	+++	+++	++	+	..	+	−	−	−
	E.n.c.	..	+	+	+	−	−	−	..	−	−	−	−
	E.n.f.	..	+	−	+	−	−	−	..	−	−	−	−
Jones	E.c.c.	..	..	++	++	+	−	−	−	−	−	−	−
	E.f.	..	..	−	−	−	+	−	−	−	−	−	−
	E.n.c.	..	..	+++	++	−	−	−	−	−	−	−	−
	E.n f.	..	..	−	−	−	−	−	−	−	−	+	+
McLaggan ..	−	−	..	−	..	−	−	−	..	−	−	−	−
Green	E.c.c.	−	..	+	+	+	−	+	+	..	+	+	..
	E.n.f.	−	..	−	−	−	−	−	−	..	−	+++	..
	E.n.c.	++	..	+	++	+	++	+	+	..	+	+++	..
Bartholomew ..	E.c.c.	−	+	++	++	+	−	−	−	..	−	..	..
	E.f.	−	++	++	−	−	++	+++	−	..	−	..	..
Cooke	−	−	−	−	−	−	−	..	..	−	−	−	..
Downs	E.c.c.	..	+	..	++	++	+	+	−	+	−	−	..
	E.h.c.	..	−	..	−	−	++	+++	+++	+++	++	−	..
	E.f.	..	++	..	−	−	−	−	−	−	+++	+	..
Jenkins	E.n.c.	..	+++	+	..	++	++	+	..	+	+	++	+
	E.n.f.	..	−	−	..	−	++	−	..	−	−	−	−
Ball	E.h.c.	..	..	..	++	++	+	+	+	−	−	−	−
	E.f.	..	..	..	−	−	−	−	−	−	−	+	−
Ballance	E.c.c.	+	+	..	−	−	+	..	..	−	+	..	−
	E.n.c.	−	−	..	−	−	−	..	..	−	++	..	−
Cherldine	E.c.c.	+++	+	+	+	+	+	+	+	..	..	..	..
	E.f.	−	−	−	−	−	+	−	−	..	..	..	..
	E.n.f.	++	−	−	−	−	−	−	−	..	..	..	..
	Tet.c.	−	−	−	−	−	++	+++	+++	..	..	..	..
	Tet.f.	−	−	−	−	−	+++	−	−	..	..	..	..
Jackson	E.c.c.	−	−	−	−	+	+	+	..	..	..	−	..
	E.h.c.	−	−	−	−	−	−	+	..	..	..	−	..
Reader	E.c.c.	+	+	−	..	..	+	−	..	−	+	..	−
	E.f.	+	−	+	..	..	−	+	..	−	−	..	+
Briggs	−	..	−	−	−	−	−	−	..	..	−	..	−
Miller	E.h.c.	..	..	−	+	..	+	+	++	++	++	++	..
Carson	E.c.c.	+	+	+	+	+	..	..	..	..	+	++	..
	E.f.	++	−	+	−	−	..	..	..	..	+	−	..
Watson, C. ..	E.f.	..	−	−	−	+	−	−	−	..	..	..	..
Watson, A. ..	E.n.c.	+	+	+	−	++	+	+	..	..	..	..	..
	E.n.f.	+	−	−	−	−	−	−	..	..	..	..	..
Hewitt	−	−	−	−	..	−	..	..	..	−	−	..	−
Cable	−	−	−	−	−	−	−	..	..	..	..	−	..

TABLE I.—*continued.*

	A	1	2	3	4	5	6	7	8	9	10	11	12
		+++										+++	
Hollow	E.f.	r.b.c.	+	+	..	..	..	..	..	+++	++	r.b.c.	+++
	Trich.	+	+	−	..	..	..	..	..	++	−	−	−
Allen	E c.c.	−	..	+	+	−	..	..	..	−	++	..	−
	E.f.	−	..	−	−	+	..	..	..	+	−	..	−
Parry	E.c.c.	−	..	..	..	..	+	..	−	−	−	−	−
	E.f.	++	..	..	..	..	−	..	+	+	−	−	−
	Tet.f.	−	..	..	..	..	−	..	−	−	−	+++	+++
Clarke	E.c.c.	+++	++	+	++	−	−	+	..	..	..	..	..
	E.f.	−	+	++	+++	+	+	+++	..	..	..	..	..
Williams	−	−	..	−	−	−	−	−	−	..	..	..	..
Draper	−	−	..	..	..	..	−	−	−	−	−	−	−
Kewley	E.c.c.	−	..	+	+	+	++	+	..	..	..	..	..
	E.h.c.	−	..	−	−	−	−	++	..	..	..	..	..
	E.f.	−	..	++	+	+	−	+	..	..	..	..	..
	L.c.	−	..	−	−	−	−	++	..	..	..	..	..
Bass	−	−	−	−	−	−	−	..	..	..	..	..	..
Bennett	−	−	−	−	−	−	−	..	..	..	..	..	..
Mullen	E.c.c.	−	..	+	+	+	+	−	..	..	..	..	..
	E.f.	−	..	−	−	−	−	−	..	..	..	..	..
	E.n.c.	−	..	++	+	+	+++	+	..	..	..	..	..
	E.n.f.	−	..	−	−	−	+	+++	..	..	..	..	..
Bond	E.c.c.	+++	++	..	−	+	−	..	..	..	..	..	..
	E.f.	−	−	..	−	+	−	..	..	..	..	..	..
	L.c.	+++	++	..	+++	+++	+++	..	..	..	..	..	..
	E.n.c.	+++	+	..	−	−	−	..	..	..	..	..	..
	E.n.f.	−	−	..	−	−	−	..	..	..	..	..	..
Sheather	E.c.c.	−	++	++	+	+	+	..	..	..	..	..	..
Haddack	E.c.c.	..	−	..	..	..	−	..	+	−	−	−	..
	L.c.	..	+	..	..	..	+++	..	+++	+++	+++	+++	..
	E.n.c.	..	−	..	..	..	−	..	++	++	++	++	..
	E.n.f.	..	−	..	..	..	−	..	++	++	−	−	..
McDonald ..	E.c.c.	..	−	..	−	−	..	−	..	−	+	..	..
	E.f.	..	−	..	−	−	..	+	..	−	−	..	..
	E.n.c.	..	−	..	−	−	..	−	..	−	++	..	..
Robertson	E.c.c.	..	..	+	..	..	−	..	+	+	..	−	−
Carr	E.h.c.	..	..	..	+++	..	+++	+	+++	+++	+++	..	..
	E.f.	..	..	..	++	..	−	+	−	+++	++	..	..
	E.n.c.	..	..	..	++	..	+	−	−	+++	+++	..	..
	E.n.f.	..	..	..	−	..	−	−	−	−	−	..	..

Bacon	E.c.c.	..	+	+	−	−	−	−	..	..	..	..	..
Robertshaw ..	E.c. c.	..	−	+	+	−	+	..	..	..	..	..	..
	E.f.	..	−	−	−	−	+	..	..	..	..	..	..
Duthie	−	−	..	−	−	−	..	..	..	..	..	..	..
Smith	E.c.c.	..	++	++	+++	+	..	..	..	..	..	..	..
	E f.	..	++	++	−	−	..	..	..	..	..	..	..
	E.n.c.	..	+++	+++	+++	+++	..	..	..	..	..	..	..
Cole	E.n.c.	..	..	−	−	−	..	..	..	..	++	−	..
	E.n.f.	..	..	−	−	++	..	..	..	..	−	−	..
Jackson	E.c.c.	..	..	..	..	..	++	−	+	+	+	..	..
	E.f.	..	..	..	..	..	−	−	−	+	−	..	..
	E.n.c.	..	..	..	..	..	−	−	−	−	+	..	..
Harris	E.h.c.	−	..	−	..	−	..	..	..	+	..	..	..
	E.f.	−	..	+	..	−	..	..	..	−	..	..	..
Ellmore	−	−	..	..	..	−	−	..	−	..	..	..	..
Carter	E.h.c.	−	−	−	..	+++	..	..	..	..	..	..	..
	E.f.	+	+	−	..	−	..	..	..	..	..	..	..
Turner	E.h.c.	−	−	−	..	+++	..	..	..	..	..	..	..
	E.f.	+	+	−	..	−	..	..	..	..	..	..	..
McClay	E.c.c.	..	++	++	..	+	+	..	..	..	..	..	..
	E.n.c.	..	++	+++	..	−	+++	..	..	..	..	..	..
Coughlin	E.c c.	..	..	+	−	−	..	..	..	++	..	..	..
	E.f.	..	..	−	+	−	..	..	..	−	..	..	..
King	E.f.	..	..	..	+	−	..	..	−	..	−	..	..
	E.n.c.	..	..	..	++	+	..	..	+	..	++	..	..
	E.n.f.	..	..	..	++	−	..	..	−	..	−	..	..
Halling	−	..	..	..	..	..	−	−	..	..	−	..	..
Hill	E.h.c.	..	..	..	..	−	..	..	..	..	++	++	..
	E.f.	..	..	..	..	+	..	..	..	..	−	−	..
Woan	E.n.c.	..	..	..	+	−	−	..	..	..	..	..	..
Evans	−	..	..	..	−	−	−	..	..	..	..	..	..
Wathnough ..	−	−	−	..	−	..	..	..	..	..	..	..	..
Melvin	E.f.	..	−	..	..	+	..	−	..	..	..	..	..
	Tet.f.	..	−	..	..	−	..	++	..	..	..	..	..
Hopkins	−	−	..	−	..	−	..	..	..	..	..	..	..

ABBREVIATIONS.

E.c.c. = *E. coli* cysts.
E.h.c. = *E. histolytica* cysts.
E.f. = Unencysted entamœbæ (either *E. coli* or *E. histolytica*).
E.n.c. = *E. nana* cysts.
E.n.f. = *E. nana* free.
L.c. = Lamblia cysts.
L.f. = Lamblia free.
Tet.c. = Tetramitus cysts.
Tet.f. = Tetramitus free.
Trich. = Trichomonas.
r.b.c. in E.f. line = entamœba with included red blood corpuscles.

Cooks of Sidi Bishr camp, 279 men.

Cooks of Mustapha Convalescent Depot, 119 men.

Cooks of camps at Mazareta, Metras, Docks, Mex and Gabarri, 191 men.

Royal Army Medical Corps staff of Orwa-el-Waska section, 19th General Hospital, 65 men.

TABLE II.—Healthy Men examined in Alexandria for Carriers of *E. histolytica* arranged with Reference to Previous History of Dysentery, Station and Service.

Camp	Where stationed	P.D.	Carriers	Per cent.	N.P.D.	Carriers	Per cent.	Total per cent.
Metras (1,013) ..	Gallipoli and Egypt	167	4	2·4	846	37	4·3	4·0
Permanent Staff, Mustapha (312)	Gallipoli and Egypt	12	0	0	47	8	17·0	13·5
	Egypt only ..	5	0	0	248	10	4·0	3·9
Sidi Bishr Cooks (279)	Gallipoli and Egypt	51	8	15·7	103	12	11·6	13·0
	Egypt only ..	4	0	0	96	5	5·2	5·0
	Salonica and Egypt	3	0	0	22	2	9·0	8·0
Mustapha Cooks (119)	Gallipoli and Egypt	9	3	33·3	26	1	4·0	11·4
	Egypt only ..	0	0	0	84	6	7·1	7·1
Mazareta, Metras, Docks, Mex and Gabarri Cooks (191)	Gallipoli and Egypt	11	0	0	72	4	5·5	4·8
	Egypt only ..	2	0	0	106	2	1·8	1·8
R.A.M.C. Staff, 19th General Hospital (65)	Gallipoli and Egypt	9	1	11·1	13	0	0	4·5
	Egypt only ..	9	0	0	34	3	9·0	7·0

P.D. = Previous dysentery. N.P.D. = No previous dysentery.

In the above table (Table II) the *E. histolytica* findings alone are set forth. The men in each group are divided into three classes according as to whether they had served in Gallipoli and Egypt, Salonica and Egypt, or in Egypt alone. The cases examined are arranged in two columns representing a previous history of dysentery or not. Each man was carefully questioned as to whether he had had dysentery, and though it is impossible to verify the men's statements or to draw a distinction between past bacillary or amœbic infections, still it is possible to obtain some idea as to the influence a previous dysentery has on the incidence of carriers of *E. histolytica*. It will be seen, at any rate, that by far the greater number of carriers gave no history of dysentery whatever. Of the healthy men who had served in both Gallipoli and Egypt there were examined 246 who gave a history of previous

dysentery and 1,137 who gave no such history. Amongst the former there were found to be 16 carriers of *E. histolytica*, giving a percentage of 6·5, while amongst the latter there were 52 carriers giving a percentage of 4·5. Of the men who had served in Egypt alone only 20 gave a history of previous dysentery and amongst them no carriers were found, but of 568 who gave no history of dysentery 26 were carriers yielding a percentage of 4·5, a result which is identical with that obtained from the similar group of men who had served both on the Peninsula and in Egypt. This fact would seem to indicate that the incidence of infection was not greater on the Peninsula than in Egypt. The men, however, who gave a history of dysentery yielded a higher percentage of carriers than those who had no such history.

The highest percentage for *E. histolytica* was found amongst cooks employed at Mustapha Convalescent Depot and at Sidi Bishr camp. The cooks of Sidi Bishr camp showed a higher percentage of protozoal infections than any other group of healthy men examined.

The total number of healthy men examined was 1,979 and the complete findings are shown in the last column of Table III. These may be taken as representative figures for the healthy troops in Alexandria, as the men were taken in groups from very different areas. It must not be forgotten that the figures are based on the result of a single examination.

TABLE III.—The Various Protozoal Infections found amongst a Series of 1,979 Healthy Troops examined in Alexandria in the Early Part of 1916.

	R.A.M.C. Staff, 19th General Hospital	Troops in Metras Camp	Permanent R.A.M.C. Staff, Mustapha Convalescent Camp	Cooks employed at Mustapha Convalescent Camp	Cooks at Mazareta, Metras, Docks, Gabarri and Mex Camps	Cooks at Sidi Bishr Camp	Total
Total examined	65	1,013	312	119	191	279	1,979
E. histolytica ..	6·1	4·0	5·7	8·4	3·1	9·6	5·3
E. coli	17·0	15·8	27·8	16·0	23·0	25·4	20·0
Entamœba (undiagnosed)	0	0·3	1·9	5·0	3·6	1·8	1·3
E. nana ..	0	0	0	1·6	0	0·7	0·5
Lamblia ..	0	4·3	5·4	6·0	7·8	4·6	4·8
Trichomonas ..	0	0·49	1·6	1·6	1·0	2·8	1·1
Tetramitus ..	0	0·39	0·6	5·0	2·6	2·1	1·1
I-cysts	4·3	2·0	3·5	2·5	2·0	6·4	3·0

The other protozoal findings in these various groups are set forth in Table III (above). It will be seen that the infections

in the various groups agree fairly closely. As was to be expected, the commonest protozoon is *E. coli*. The second is *E. histolytica*, while lamblia comes next. Tetramitus and trichomonas occurred in equal frequency. Undiagnosed entamœbæ (unassociated with cysts and not including red blood corpuscles) are placed in a group apart. The iodine-cysts were fairly regularly encountered, as were the small amœbæ which we have called *Entamœba nana*.

(*b*) *Convalescents*.—It was thought that some insight into the various protozoal infections would be gained by examining convalescents from various diseases in the Convalescent Depot at Mustapha. Accordingly, we examined 328 convalescents, with the results set out in the second column of Table IX. It will be seen that the percentages of infections did not differ to any extent, though they were generally higher than those we found amongst the healthy men, and, furthermore, they resembled very closely the findings made by one of us in London last year amongst 556 cases which had been invalided from Gallipoli and the Eastern Mediterranean (Table IX). It will be seen that the London cases gave slightly higher percentages all round. Another notable feature is the absence of any coccidial infections amongst the Egyptian cases. Apart from the difference in the coccidium infections, the variation in the two results can be explained by the fact that many of the London cases were examined more than once, and infections not found on first examinations were encountered at subsequent ones. From what has been said above regarding the fallacy attending the single examination, it is perhaps surprising that the results agree as closely as they do. The Mustapha cases were only examined once.

TABLE IV.—The Carriers of *E. histolytica* amongst a Series of 328 Convalescents in Mustapha Convalescent Depot, Alexandria, arranged with Reference to Stations and Past Dysentery.

Where stationed	P.D.	Carriers	Per cent.	N.P.D.	Carriers	Per cent.	Total per cent.
Gallipoli and Egypt ..	58	6	12·0	159	11	7·0	7·8
Egypt only, or Egypt and Salonica	8	0	0	103	4	4·0	4·0

Of these convalescents, 217 had served on the Peninsula, and of this number 58 gave a history of previous dysentery, while 157 had no such history. The percentage of *E. histolytica* carriers

amongst the former was 12·0, while amongst the latter it was 7·8. There were 111 convalescents who had not been on the Peninsula, and they had all served in Egypt alone except 11 who had been at Salonica. Only 8 gave a previous history of dysentery, and no carriers were found amongst them, while the remaining 103 who had no history of dysentery yielded 4 carriers. These results are shown in Table IV.

TABLE V.—The Result of the Examination of the 961 Hospital Cases arranged according to the Character of the Stool (in Percentages).

	Formed	Unformed	Liquid	Blood and mucus	Total
Total cases	140	393	263	165	961
E. histolytica cysts	3·0	3·3	1·1	0·6	2·2
E. histolytica free (r.b.c.) ..	—	—	—	6·1	1·0
E. coli cysts	6·2	14·0	12·0	3·0	10·4
Entamœba (undiagnosed) ..	—	3·8	1·9	—	2·0
E. nana	0·7	5·0	5·0	0·6	3·0
Lamblia	2·8	10·0	6·0	3·0	6·0
Trichomonas	—	3·6	5·7	—	3·0
Tetramitus	—	3·6	3·0	3·0	2·8
I-cysts	—	0·2	0·4	0·6	0·3

(*c*) *Hospital Cases.*—Those included under this heading are cases which were admitted to the Orwa-el-Waska section of the 19th General Hospital for dysentery, diarrhœa, and other intestinal disorders. In practically all these cases the entire stool was examined, and the character of the stool noted. The great majority of those described as blood and mucus were from cases of bacillary dysentery, while only a few (6·1 per cent.) were from cases of amœbic dysentery. In Table V given above the findings in the 961 cases tabulated are arranged according as to whether the stool was formed, unformed, liquid, or blood and mucus. In the latter case it does not necessarily mean that the stool consisted of nothing but blood and mucus, but that blood and mucus were present. In the majority, however, as these were cases of bacillary dysentery, blood and mucus were alone present, or formed the bulk of the stool in the manner characteristic of this disease. The percentages in the four columns show clearly that protozoa were most common in the unformed or liquid stools. It is, perhaps surprising that the encysted forms of *E. coli* and *E. histolytica* were more often found in stools of this kind than in the formed stool. As was to be expected, free forms of *E. histolytica* with included red blood corpuscles were only found in the case of the blood and mucus

stools yet encysted; *E. histolytica* were found fairly frequently in this type of stool. (In an examination made subsequently to the tabulating of these results, an active entamœba with included red blood corpuscles was found in a stool which was unformed, and which contained no blood or mucus either macro- or microscopically. Similarly trichomonas was found on one or two occasions in bacillary dysentery stools consisting of nothing but blood and mucus examined after the table was completed.)

If we compare these results obtained from hospital cases mostly admitted for intestinal disorder with the findings in healthy men and in convalescents (*see* Table IX, *page* 26), the most striking feature is the low percentage of *E. histolytica* and *E. coli* infections, while the flagellate infections are higher, especially in the case of the unformed and liquid stools. This does not necessarily mean that the flagellates are the cause of the unformed or liquid stools, though they may be in some cases. With trichomonas, and to a lesser extent with tetramitus, which has a recognizable encysted stage, there is a difficulty of recognition in the formed stool. It is almost certain that if the stools of the healthy men, which are mostly formed when examined, were rendered liquid by the administration of salines, then the percentage of recognized flagellate infections would be increased. This is borne out by the fact that in the case of the *E. histolytica* carriers which were brought into hospital for treatment, flagellate infections were only recognized in many cases after the patients had been treated with salines. An examination of the charts produced at the end of this paper, and giving the histories of these *E. histolytica* carrier cases, will show how frequently the flagellates appear as the stools become soft. All the cases referred to, it must be remembered, were carriers of *E. histolytica*, which were found during the course of routine examinations of healthy men.

As we have already remarked, the majority of the 165 cases showing blood and mucus were undoubtedly cases of bacillary dysentery.* Definite amœbæ with included red blood corpuscles were found in only 6·1 per cent. of these cases. The remaining 93·9 per cent. of cases were probably of a bacillary nature, making bacillary dysentery over sixteen times more frequent than amœbic

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* About sixty of these cases were plated for culture of dysentery bacilli. In over half of these *B. dysenteriæ* either Shiga or Flexner was isolated. This gives a percentage of success which is quite up to the average for the isolation of *B. dysenteriæ* from cases of this disease.

dysentery. This was quite in agreement with the experience of other hospitals in Egypt during the period covered by this report. Bacillary dysentery was everywhere much more common than amœbic dysentery, which was a comparatively rare disease in spite of the fact that such large numbers of carriers of *E. histolytica* existed.

(*d*) *British Prisoners in the Military Prison, Gabarri.*—An examination was made of 168 prisoners in the military prison. The findings in this case are of interest, for it was noted that the majority of the men were suffering from some intestinal disorder—the stools of as many as 138 of the 168 men being abnormal in one way or another. A bacteriological examination made by Captain Campbell, R.A.M.C., bacteriologist to the 19th General Hospital, of the stools of ten cases taken at random yielded definite dysentery bacilli (mannite fermenting) in three, and bacillus Morgan No. 1 in four. The protozoological findings which are shown below did not afford any explanation of the condition of the men. Many of the men were passing dysenteric stools, others had chronic diarrhœa, and others were passing abnormally loose motions.

The protozoa found are distributed very much as they are amongst the cases examined in hospital (Column 5, Table IX, *page* 26). The *E. histolytica* infections are lower, but the figure 1·8 should most certainly be increased at the expense of the 17·2 for the undiagnosed amœbæ. It is noteworthy that the flagellate infections amongst these men are almost identical with those found amongst the hospital patients. The *E. nana* infections were higher than in any other group examined. The percentage of cases infected with blastocystis is high (41·0), but it was only in this group that a special note of the occurrence of this organism was made.

Though the men in Gabarri Prison were supposed to be healthy men, the condition of their stools showed them to be otherwise, with the result that they were more in line with the cases admitted to hospital for dysentery, diarrhœa, or other intestinal trouble.

Amongst these men again, as with the hospital cases, the general looseness of the stools explains the higher percentage of flagellate infections.

It is, perhaps, worthy of note that a very large proportion of the prisoners showed in the stool a large spore-bearing bacillus. This was found in at least 30 per cent. of the cases examined, and was frequently present in such numbers that the microscopic field was

covered with the bright refractile spores. The bacillus was isolated, and Captain Campbell, R.A.M.C., identified it as *Bacillus megatherium*. It was often present in large numbers in stools containing mucus, the mucus itself appearing to be a very favourable medium for its growth. When amœbæ were present, the spores of the bacillus were frequently ingested by them.

(*e*) *Healthy Natives.*—It was evident as a result of examinations made by one of us in England during the latter part of 1915, and by both of us in Egypt, that the British troops in the Mediterranean war area were becoming heavily infected with various intestinal protozoa. We have shown that this infection is probably largely due to the flies which readily take up these protozoa from human excrement and deposit them upon articles of food. It is evident that for such an infection to be possible a reservoir must exist, and the native seemed the most probable source of this infection.

In order to test this theory it is necessary to examine the natives of Egypt, and we were able to do this owing to the kindness of Dr. Kirton, who gave us permission to examine a number of prisoners in Hadra Prison, Alexandria. We examined in all 524 prisoners, with the results set out in Column 6, Table IX, *page* 26. As the helminthic infections were so common in these men we have thought it worth while to include them also (Table VI.)

TABLE VI.—Helminthic Infections amongst 524 Healthy Native Prisoners in the Hadra Prison, Alexandria.

	No.	Per cent.		No.	Per cent.
Trichocephalus ..	68	12·9	*Tænia saginata* ..	21	4·0
Ascaris	265	50·4	*Tænia solium*	3	0·57
Ankylostoma ..	55	10·5	Bilharzia	19	3·6
Oxyuris	5	0·98	*H. heterophyes* ..	2	0·38

It will be noted that the *E. coli* and *E. histolytica* infections are high while the flagellate infections are low. The I-cyst infections are also high. As the results are all based on a single examination it is evident that the infections are really considerably higher than our figures indicate. Nevertheless, the percentages are high enough to justify the conclusion to be drawn. As many as 13·5 per cent. of the men were infected with *E. histolytica* and 48·6 per cent. with *E. coli*. The I-cyst infections were even a little higher than the *E. histolytica*. It is evident, therefore, that the number of healthy natives infected with *E. histolytica*, *E. coli* and

I-cysts, is sufficient to account for the spread of these infections to British troops in and around Alexandria, especially when we remember the part played by flies in the transportation of these protozoa and the manner in which the natives deposit their fæces broadcast over the land.

We examined a certain, though small, number of actual human fæcal deposits collected from corners and open spaces in Alexandria and found, as was to be expected, that the percentages of infections were not lower than those obtained amongst the prisoners in Hadra Prison. It is evident, therefore, that the native carrier is the source of infection for *E. histolytica* and other intestinal protozoa. The scarcity of flagellates amongst these natives has already been mentioned, and it may be that in the case of the flagellates some other reservoir exists, possibly some animal associated with man. It is noteworthy that we have found both trichomonas and lamblia in cats in Alexandria, while these flagellates are known to occur in rats and mice. So far no proof has been obtained as to the identity of these with the parasites of the human intestine.

To return to the prisoners of Hadra Prison, it is interesting that very few indeed of these men had any sign of intestinal disorder. They had a uniform diet, and this had the effect of producing a curious uniformity and monotony in the character of the stool. In the case of only three of the men could the stool be described as abnormal, and in one of these, a soft unformed stool, tetramitus was found.

A classification of the prisoners examined was made according to the length of time they had been in prison. It was found that the infections amongst those who had been in jail one week only or under this time were not lower than amongst those who had served longer periods. It was evident that the infections could not be the result of life in jail—in other words, that the infections were not jail infections. This is borne out also by the result of our examination of fæcal deposits taken at random about the town which has been referred to above.

(*f*) *Native Cooks.*—In addition to the examination of native prisoners in Hadra Prison, we examined a series of eighty-seven native cooks who were employed in an Army bakery, and the Hotel Metropole, which was used as a restaurant by soldiers employed in Alexandria. The natives were of a better class than the native prisoners in Hadra Prison, and their infections conform more to the type found amongst the British troops. The amœbic infections

were lower, and the flagellate infections higher than among the prisoners. The results of this examination are shown in Column 7 of Table IX, *below*.

The helminthic infections were also lower than amongst the native prisoners.

TABLE VII.—Helminthic Infections amongst Eighty-seven Native Cooks in Alexandria.

	No.	Per cent
Ascaris	3 ..	3·4
Ankylostoma	3 ..	3·4
Trichocephalus	3 ..	3·4
Strongyloides	1 ..	1·1

(*g*) *British West Indian Troops.*—Only forty-eight of these natives of British West India were examined, and they were all employed as cooks at Mex Camp, where the British West Indian troops were stationed. The protozoal infections give figures which are considerably lower than those of the natives of Alexandria examined in the Hadra Prison, and resemble more the results we obtained with the British troops (Column 7, Table IX, *below*). The helminthic infections were, however, high, and we have added our findings in this direction. These results are shown in the following table :—

TABLE VIII.—Helminthic Infections amongst Forty-eight British West Indian Soldiers in Alexandria.

	No.	Per cent
Trichocephalus ..	15 ..	31·2
Ascaris	12 ..	25·0
Bilharzia (sc. h.) ..	1 ..	2·0
Ankylostoma	8 ..	16·0
Strongyloides	1 ..	2·0

TABLE IX (Composite Table).—Protozoa found amongst Convalescents and Healthy Troops in Alexandria and London and Natives in Alexandria. (Percentage of Infections).

	Healthy troops	Convalescents Alexandria	Convalescents London	Hospital cases	Gabarri Prison	Hadra Prison. Natives	Native cooks	British West Indian cooks
Total examined ..	1.979	328	556	961	168	524	87	48
E. histolytica ..	5·3	6·4	10·8	3·2	1·8	13·7	11·5	4·1
E. coli	20·0	31·7	39·0	10·4	12·0	48·6	20·7	18·7
Lamblia	4·8	5·4	16·0	6·0	6·0	0·57	7·0	4·1
Trichomonas ..	1·1	0·67	1·6	3·0	2·4	0	1·1	0
Tetramitus	1·1	0·9	0·7	2·8	3·2	0·19	1·1	0
Coccidium isospora ..	0	0	2·7	0	0	0	0	0
Coccidium eimeria ..	0	0	0·2	0	0	0	0	0
Entamœbæ (undiagnosed)	1·3	1·8	0	2·0	17·2	0·57	1·1	0
Iodine cysts	3·0	2 0	5·2	0·3	0	14·8	7·0	4·1
E. nana	0·5	0	1·0	3·0	12·0	0	0	0

In the case of the London convalescents who were examined in London by one of us (C. M. W.) during the latter part of 1915, the small amœba now entered as *E. nana* was in the original report entered as *Amœba limax*. As will be explained below this diagnosis was most probably erroneous. Coccidium (isospora) was found once, and the flagellates *Tricercomonas intestinalis* and *Waskia intestinalis* several times in Alexandria, but as they were not found at the first examination of the cases, they are not included in the table.

General Remarks on the Protozoal Infections.

The results of the examinations of the various groups described above are set out in tabular form in Table IX.

(1) *Entamœba coli.*—It will be seen at once that *E. coli* is by far the commonest protozoon of the human intestine in Egypt as in all other countries where it has been sought. Amongst the British troops it was found most commonly in the case of the convalescents, 31·7 per cent. of those examined showing this parasite. The convalescents from the Eastern Mediterranean examined in London during the latter half of the year 1915 showed a similar high percentage of *E. coli* infections. It is difficult to explain why the convalescents (mostly from dysentery or other intestinal disorders) should show a higher percentage than healthy men of a non-pathogenic amœba like *E. coli*. It is possible that a damaged intestine is more liable to become infected with this protozoon, though why this should be so it is difficult to imagine.

The high percentage of *E. coli* infections amongst the natives of Alexandria has already been referred to above. It is interesting to note that the forty-eight British West Indians examined show a lower percentage of infections than any other group of healthy men examined. The low figure in the hospital cases and the men in Gabarri Prison, who were mostly suffering from some intestinal disorder, is to be explained by the soft or liquid character of the stool, as has been noted above. If a series of diarrhœic cases are examined it can be stated, as a rule, that the figures for the amœbæ will be below the average for healthy men, while those for the flagellates will be above.

(2) *Entamœba nana.*—The small amœba which we have called *E. nana* was found to be a common parasite in Egypt. It was some time before we were able to identify the cyst of this amœba, for in the first two or three months we were accustomed to regard

it as of a vegetable nature. It was for this reason that the amœba was not noted in any of the natives in Hadra Prison in the 197 healthy men, nor in the 328 convalescents. The amœba was certainly present but was neglected by us when in the encysted state, or was regarded as a small form of *E. coli* or *Amœba limax* when free. In the other groups examined subsequently it was constantly present, and attained its highest percentage in the case of the men in Gabarri Prison. As will be explained below, the small amœba identified as *A. limax* in the case of the London convalescents, was in all probability *E. nana.*

(3) *Coccidium* (*Isospora*).—Infection with this coccidium was only met with once in the case of an *E. histolytica* carrier who was being controlled in hospital. The oöcysts were first noted about a week after the beginning of the observation. It is remarkable that fifteen cases of this infection were met with in London out of 556 cases examined, while in Egypt only one was seen out of a much larger number of examinations. It seems that coccidium infections were fairly common in Egypt during the latter part of 1915 when they were being found in England, and this may be explained by the fact that the men in both places had then come recently from Gallipoli where the infection may have been acquired. The scarcity during the first six months of 1916 in Egypt may be due to the fact that the infection had in most cases died out. There may, however, be some seasonal incidence of the infection. As the figures in the tables are based on the single examination the one case of coccidiosis does not appear, as it was only found at a later examination.

(4) *Entamœba histolytica.*— One of the main objects of the investigation we undertook in Alexandria was the determination of the percentage of carriers of *E. histolytica* amongst healthy troops. From what has already been stated and the figures shown in Table IX, it will be seen that there is a high percentage (5·3) of carriers amongst the healthy British troops in Egypt. The figure would be higher if allowance were made for the error of the single examination on which the figure 5·3 is based. The carriers were no more numerous amongst men who had served in Gallipoli, as we have already shown above. This fact would seem to indicate that Gallipoli was not more heavily infected with *E. histolytica* than Egypt, which has been known to be an endemic centre for amœbic dysentery for upwards of forty years.

The convalescents examined in London and Alexandria showed a higher percentage of carriers than the healthy men, while the

hospital cases, on account of their frequently liquid stools with their flushing properties, gave a much lower figure. It should be remarked here that many of the hospital cases which gave a negative result when the stool was liquid gave a positive result later when the stools became formed. As the figures are those resulting from the single examination, the findings of subsequent examinations were not included.

The presence of such a large number of carriers amongst healthy men was not suspected, and came as a somewhat startling revelation. The percentage was highest amongst the British troops in the case of cooks employed at Mustapha and Sidi Bishr camps. It is difficult to explain why this should be so unless the flies which swarm about the cook-houses are sufficient to account for it. The origin of all these infections is undoubtedly the native, as shown by the high figure obtained in the case of the prisoners in Hadra Prison.

The carrier problem of *E. histolytica* raises many important questions which will be dealt with below.

(*a*) *The Possibility of examining Healthy Troops, with a View to the Isolation of Carriers.*

Provided there were means at our disposal for the separation of all carriers, and that the examination did not involve any great delay, it would undoubtedly be advisable to separate and treat with emetin all carriers of *E. histolytica*. At the present time, however, such a course is absolutely out of the question, for we not only have no means for undertaking examinations on such a scale—examinations which are often difficult even for a practised observer—but it would be quite unjustifiable to detain such a large number of healthy men for the time required for examination and treatment. Even supposing that all the carriers amongst the troops were separated and treated in a country like Egypt, we are no better off, for the source of infection is always present in the shape of the native and the fly, with their insanitary habits. In the Alexandria district the course was adopted of examining all the cooks employed. This involved a great amount of laborious work which was admittedly incomplete, as only a single examination was made. Still, this was a distinct advantage, as all the carriers which were thus identified were separated, and most of them were cured by emetin treatment. There was nothing, however, to prevent the men re-infecting themselves, and in order permanently to keep down the number of carriers amongst any group of men it would

be necessary to exàmine them regularly at stated intervals. It seems, therefore, that in a country like Egypt very little can be done in the way of isolating carriers of *E. histolytica* from amongst healthy troops, and one could not possibly advise that it should be undertaken on a large scale in time of war.

(*b*) *The Danger of Amœbic Dysentery spreading in England.*

Another aspect of the question is the possibility of the spread of amœbic dysentery to countries in which the disease does not already exist, and it has been suggested that troops which are to be moved from an area where the disease is endemic should be examined and the carriers detained. Here again the amount of work which would be necessary to carry out such a project shows this to be quite impossible in time of war except in the case of small drafts. But in a country like England is there any real danger that the disease will be spread in this manner? Cases of amœbic dysentery contracted in England are not unknown, though they are far from common. It seems very improbable that the disease will establish itself there, for though it is only recently that the carrier problem of amœbic dysentery has attracted attention, it must not be forgotten that carriers have been constantly entering England before the present War, and that troops have often returned from countries in which the disease is endemic. There have undoubtedly been large numbers of carriers amongst such men, though no one has considered it worth while to examine them from this point of view. There must have been in the past every possibility that the disease would establish itself in England if the conditions favourable for the survival of cysts outside the body and their transference to other individuals had existed. Though isolated cases of infection have occurred, the disease has been exceedingly rare. This failure of the disease to establish itself must be the result of many factors, the most important of which are undoubtedly the existence of a good sanitary system and the comparative absence of flies, while climatic conditions undoubtedly play a prominent part. In England, even in rural districts where the sanitary arrangements are often far from perfect, the infected material is not spread broadcast over the land as it is in countries like Egypt ; while flies, though very numerous in certain localities, are never so universal as they are in warm countries, which are the natural homes of the disease.

With the establishment of large camps in rural districts of England, we may expect some temporary increase in the local cases

of amœbic dysentery, but the disease is unlikely to gain a permanent footing in the country, for the British carrier will never in the long run aid the spread of the disease as does the insanitary native of Egypt.

(c) *The Advisability of examining all Convalescents with a View to the Isolation of Carriers of E. histolytica?*

With such a high percentage of *E. histolytica* carriers amongst healthy men in Egypt, it is evident that at least the same percentage of carriers will be found amongst convalescents. This has actually been the case amongst men who have been invalided not only for various intestinal diseases, but for quite other conditions. Men convalescing from many different diseases are constantly being invalided from Egypt, Mesopotamia, and other areas in which amœbic dysentery is endemic. The great majority of these men when they reach England have recovered from their illnesses, and many of them are able to return to duty. A certain number, however, are still ill. In the case of those men who have recovered, it would seem quite unnecessary to institute examinations with a view to isolating the carriers of *E. histolytica*. These men are in the position of the healthy troops who are still on full duty, and amongst them the carriers are almost, if not quite, as numerous. If these men are clinically fit to return to duty, there does not seem any just reason for detaining them longer, for they will in no way increase the percentage of carriers amongst the healthy troops who have been removed from areas in which amœbic dysentery is endemic. In our opinion there is at the present time no justification for detaining a man just because he has been accidentally found to be a carrier of *E. histolytica* in the course of routine examination, even though he was invalided for dysentery in the first place.

With men, however, who are ill, whether their condition leads one to suspect that *E. histolytica* is or is not the cause of their illness, the question is a different one. Such cases can be suitably treated with emetin if *E. histolytica* is present. In these cases there would be no unnecessary detaining of healthy men.

It seems to us, therefore, that in dealing with convalescents at the present time from the point of view of *E. histolytica* infections the test should be a clinical one. If the men are clinically fit they should be discharged to duty, whereas if they are not fit an *E. histolytica* infection should always be looked for and treated, for it may be the cause of trouble, and involves no unnecessary

detention of men who would be otherwise returning to duty. It is not of vital importance at the present time that all convalescents any more than all healthy troops should be examined for *E. histolytica* infections. The quick return to duty is far more important. Examination is only necessary in the case of men who are still ill and unable to return to duty. A man should be regarded as clinically fit if he shows no sign of the disease from which he suffered, is able to take full diet and undertake light duty and at the same time passes a normal stool, that is to say a stool which is macroscopically normal. It seems to us quite unnecessary and a waste of time to examine the healthy stools of such men with the object of detecting *E. histolytica* or other protozoal infections, for the finding of *E. histolytica* cysts in these men does not mean that they have had or are soon likely to have amœbic dysentery any more than the healthy men who have not been invalided. Further, although we fully recognize that the convalescent cyst carrier may be a source of danger to others, the same is true of the much larger number of healthy and unsuspected cyst carriers whom it is, of course, quite impossible to detect, far less examine.

(*d*) *The Length of Control Necessary after Treatment.*

As there cannot be any question at the present time of eliminating all the *E. histolytica* carriers from amongst the troops it is unnecessary to control any healthy carriers which have accidentally come to light, after the completion of a course of treatment. As will be shown in the section devoted to the treatment of healthy carriers, it is possible to cure the infection in practically every instance by the combined subcutaneous and oral administration of emetin. *This refers only* to the *healthy* carriers, the great majority of whom have no history of dysentery. When a history of dysentery has been obtained it is in most instances impossible to decide whether it was bacillary or amœbic. There are, however, unhealthy carriers. These are cases which suffer from chronic amœbic dysentery. They have repeated attacks when blood and mucus appear in the stool along with active amœbæ with included red blood corpuscles. Alternating with these are periods when dysentery is not evident and the stools may or may not be normal, though generally they are soft and mucoid. Microscopic examination at this time reveals the same condition (cysts and free amœbæ) as in the healthy carrier. These chronic amœbic dysenterics are most difficult to cure and, as will be shown below, the majority relapse

sooner or later. Such cases are quite incapable of going back to duty and they have to be watched carefully for some weeks after any course of emetin treatment, for the latter is very misleading in giving a sense of false security, as in nearly every case it brings about a *temporary* cure.

In dealing with convalescents from dysentery or other intestinal disorders in war time we would, therefore, suggest the following rules :—

(i) If the case has clinically recovered and is able to take full diet and perform light duty and passes a normal stool, it is not necessary to submit the stools to microscopic examination for the detection of *E. histolytica* or other protozoa. Such cases if examined would yield a certain number of *E. histolytica* carriers, but these would be hardly more numerous than the carriers amongst a group of healthy men who have not been invalided. These men can return to full duty.

(ii) If the case has not recovered, being still ill or passing abnormal stools, microscopic examination should be carried out and any *E. histolytica* infection treated, as this may be the cause of the trouble.

(iii) Supposing, however, it is decided to isolate and treat all the carriers amongst the dysentery convalescents, this is not to say that it is necessary to control the healthy carriers after treatment. In nearly every instance the treatment as explained below will lead to a permanent cure, and it is only a waste of time to detain for purposes of control men who were not suffering from their infection even when it was present. In the case of men who have suffered from repeated attacks of amœbic dysentery and are chronic amœbic dysenterics a careful control of at least one month after treatment is complete is indicated. As will be shown below the treatment of such cases is very unsatisfactory.

(5) *Flagellate Infections.*—As regards the flagellate infections the most noticeable feature is the fact that the highest percentages found were amongst the hospital cases and the men in Gabarri Prison. As we have already explained, these two groups resembled one another as regards the character of the stools. It might be urged that this is an argument in favour of the pathogenicity of the flagellates, but it seems more probable that the flagellates have become evident because the stools are liquid or soft. Evidence in this direction is to be obtained from the *E. histolytica* carrier cases which were taken into hospital for treatment and were examined every day. These men were to all intents and purposes

healthy men passing normal stools. During the course of treatment, on account of the administration of salines or emetin, the stools became liquid or unformed, and with this change flagellate infections which had not been evident in the formed stools often made their appearance. It will be noted that in the tables the two flagellates, Tricercomonas and Waskia, which are described on *pages* 86–90, do not appear. This is due to the fact that though they were found in carriers of *E. histolytica* it was not at the first examinations when the stools were normal but only later in the observation when the stools had become soft. It seems quite clear that the percentage of flagellate finds in a group of healthy men would be considerably increased if the stools were first rendered liquid or soft by the administration of salines for a few days.

The Question of invaliding for Flagellate Infections.

At the present time, there are being admitted to the hospitals in many of the war areas cases of intestinal disorder which are associated with flagellate infections of the intestine. Many of these cases are finding their way to England and others are being discovered there, and it becomes a matter of importance to decide whether such infections are to be regarded in themselves as a sufficient cause for the invaliding of a man. Microscopic examination of the stool for protozoal infections has never been undertaken before to the extent it has reached in the present War. Formerly this branch of examination was completely neglected and it is only during the last year or so that the subject has attained any importance. But very few, even of those who have taken an interest in tropical diseases and have been accustomed to teach this branch of medicine, have had any previous knowledge of the subject apart from the fact that amœbæ produce a form of dysentery and that the flagellates may be found in diarrhœic conditions. Since the commencement of the War, however, the interest in the intestinal protozoa of man has extended very much, and many microscopists have taken up the study of these protozoa and are able now to differentiate between the various intestinal amœbæ and flagellates of man.

We think there is a danger that undue importance will be attached to the mere presence of protozoa in the intestine. It is an undoubted fact that from the point of view of efficiency the vast majority of men showing protozoal infections are quite normal and capable of undertaking their regular duties. This is true even of

the majority of carriers of *E. histolytica*, a protozoal organism which may lead to most serious consequences.

It seems to us that the mere fact that a man is found in the course of routine examination to be infected with lamblia, trichomonas or tetramitus, is no justification for certifying him as a carrier who must be isolated and treated. We recognize that a certain small percentage of men showing these infections are ill, but quite apart from the possibility of the flagellates being the cause or the only cause of the malady from which they suffer, these men must be invalided on clinical grounds, and treatment may then be directed against their flagellate infections if these are thought to be the cause of the trouble. The test, therefore, in our opinion, of every case of flagellate infection must be a clinical one, and so soon as any man's symptoms clear up, even though the flagellates lamblia, trichomonas and tetramitus are still present, or are known to have only temporarily disappeared from the stool, the man should be discharged to duty. Unless this rule is followed very soon, the hospitals and convalescent camps will become filled with men who are quite capable of performing their duties as good soldiers.

It does not seem to be thoroughly realized that as there is a normal bacterial flora of the human intestine so there may be a normal protozoal fauna. Just as the majority of the intestinal bacteria never cause any trouble whatever, so most of the intestinal protozoa live in the gut without doing any harm and without producing any symptoms. If this fact was properly understood there would be less risk of every protozoal infection being regarded as a source of danger.

The Dysentery of the Eastern Mediterranean in 1915.

It is now universally known that dysentery was the cause of invaliding of large numbers of our soldiers from the Peninsula in 1915 and the impression gained ground that, at any rate during the first few months of the campaign, the dysentery was mostly of the amœbic type. It will be admitted that the epidemic was quite unexpected, and at first arrangements for adequate diagnosis of the cases did not exist, so that all the errors which will be discussed in this paper must inevitably have crept in. In the majority of cases, those who had to do with the diagnosis had very little or no previous experience of the intestinal protozoa of man, and were unconcious of the difficulties to be encountered in distinguishing between pathogenic and non-pathogenic entamœbæ, or even between

entamœbæ and some of the large macrophages and other cells encountered in bacillary dysentery infections. It seems certain, therefore, that the prevalence of amœbic dysentery must have been considerably exaggerated.

That amœbic dysentery did actually occur is an undoubted fact, for Captain Archibald, who had had considerable previous experience of the disease, met with it fairly commonly in his laboratory at Mudros. Further, many of the cases were clinically of the amœbic rather than the bacillary type, as Captain Campbell who was working at Cape Hellas informs us. Evidence in another direction is obtained from the results of one of us (C. M. W.) who examined in London a large number of cases invalided from the Peninsula in the latter part of 1915. Amongst these cases there was a percentage of over ten of carriers of *E. histolytica* and protozoal infections were generally high. These men had mostly come direct from Gallipoli, so that *E. histolytica* infections must have been common on the Peninsula. Captains Archibald and Hadfield, working at Mudros East, state (*Journal of the Royal Army Medical Corps*, June, 1916) that of 518 dysenteric stools examined 362, or seventy per cent., were due to amœbic infections. The authors in explaining their method of diagnosis state that entamœbæ containing phagocyted erythrocytes were regarded as pathogenic, a view with which we entirely agree, but they do not assert that all their cases were diagnosed on this basis, nor do they tell us in what proportion of their cases the non-pathogenic *E. coli* was found. Furthermore, they state that the evidence was obtained by the direct examination of the amœbæ or their cysts. Now it would be interesting to know what was the number of cases diagnosed by the occurrence of cysts in the stool. As will be explained below, the presence of the cysts *E. histolytica* in the stool, though it proves infection with this amœba, does not necessarily mean that the case is or has been one of actual amœbic dysentery. Other forms of dysentery occur in cases which are carriers of *E. histolytica*, and in such there is produced a dysenteric stool in which cysts of *E. histolytica* may be found. The section of the above report by Captain Campbell on the work done at Cape Hellas shows that sixty-five per cent. of the stools with blood and mucus contained amœbæ, but he says definitely that phagocytosis of red blood corpuscles was noted at times. It is evident, therefore, that in many cases the amœbæ found by him must have been *E. coli*, which, judging by the results obtained in London, were more than three times as common as *E. histolytica* in men on the Peninsula.

In other laboratories in the Eastern Mediterranean, cases of amœbic dysentery were met with, but none of these with whom we have discussed the subject are willing to admit that amœbic dysentery was present to the extent that has been made out. Captain Campbell tells us that the conditions were not necessarily the same all over the Gallipoli war area. He suggested that foci of amœbic infection might exist and that in this manner discordant results would be obtained.

The results obtained by Ledingham, Penfold and Woodcock, at the King George V. Hospital in London (*British Medical Journal*, November 13, 1915), throw some light on this question. Cases returning from Gallipoli were examined both bacteriologically and protozoologically. In one series representing cases which had left the Peninsula, in June, July and August, dysenteric stools occurred in fifteen cases and dysentery bacilli (chiefly Shiga) were recovered from all of these, while *E. histolytica* occurred in none. This result is difficult to explain if it is assumed that amœbic dysentery was more prevalent than bacillary during the first months of the campaign. In a later series of cases these observers found by the agglutination test that 47·5 per cent. gave evidence of past bacillary dysentery, while none were amœbic. Even allowing for a possible reduction of amœbic infection by emetin, it would seem that bacillary dysentery was more common on the Peninsula during the early months of 1915 than has been supposed.

The general character of the outbreak on the Peninsula in its epidemic form is so contrary to what we know of amœbic dysentery that one hesitates to ascribe the bulk of the dysentery to the *E. histolytica*. It seems more probable that many factors were at work, some of them not yet identified. We feel, therefore, that there is a tendency to over-estimate the amount of amœbic dysentery on the Peninsula during the summer months of 1915, though we recognize that this disease undoubtedly was an important factor in the invaliding of our troops from this section of the Mediterranean area.

Summary of Matter discussed in Part I.

(1) The collection of samples of stools from healthy men in camps for purposes of bacteriological and protozoological examination can be successfully carried out, provided a definite system is established. Such a method, which yielded all that was required, is described in the text.

(2) The examination of a single sample from any individual for intestinal protozoa gives a result which is far from reliable. In a series of cases examined with a view to the discovery of the error, the number of *E. histolytica* infections resulting from repeated examinations were three times as great as the result obtained at the first examination. If, however, the protozoal organism is the actual cause of any intestinal trouble at the time of examination it is usually present in large numbers and is rarely missed on the first examination.

(3) Amongst 1,979 healthy men in camps 106 were found to be carriers of *E. histolytica*, giving a percentage of 5·3. Of these, 1,383 had served on the Peninsula as well as in Egypt, and of them 246 gave a history of previous dysentery, giving a percentage of carriers of 6·5, while 1,137 gave no such a history and there was only a percentage of carriers of 4·5. Of 568 men who had served only in Egypt, and who gave no history of previous dysentery, the percentage of carriers was the same, namely, 4·5.

(4) Amongst the 1,979 healthy British troops examined in Alexandria, the percentage of protozoal infections generally was lower than amongst convalescents from dysentery and other intestinal troubles examined in Alexandria and London.

(5) The commonest protozoan found in healthy British troops in Egypt was *E. coli* (20 per cent.), *E. histolytica* came next (5·3 per cent.), and then *Lamblia intestinalis* (4·8 per cent.). *Trichomonas intestinalis* and *Tetramitus mesnili* were found with equal frequency (1·1 per cent.). *E. nana*, a new entamœba, was fairly common (0·5 per cent.), while iodine cysts occurred frequently (3·0). These figures are all subject to the error involved in the single examination method.

(6) An examination of 328 convalescents from various diseases (chiefly dysentery or other intestinal disorder) in the Mustapha Convalescent Depot gave percentages of infections very similar to those obtained in the examination of a similar series of cases in England in 1915. The percentages were generally higher than amongst the healthy troops.

(7) An examination of 961 cases admitted to the Orwa-el-Waska Section of the 19th General Hospital between January and July, 1916, showed a lower percentage of *E. histolytica* and *E. coli* infections than amongst the healthy and convalescent men in camps. The flagellate infections (lamblia, trichomonas and tetramitus), however, were higher. This is explicable on the

ground that many of the cases were suffering or had just suffered from diarrhœa, a condition which tends to get rid of amœbic infections (temporarily) but tends to reveal a flagellate infection which is not easily detected in the formed stool.

(8) An examination of 524 healthy natives in Hadra Prison, Alexandria, showed a high percentage of infection with *E. histolytica* (13·5), *E. coli* (48·6) and I-cysts (14·8). The flagellate infections were low. In only three of the 524 cases could the stool be described as abnormal. Similar results were obtained in the examination of human fæcal deposits collected in and around Alexandria.

(9) It is evident that the native of Egypt is acting as a reservoir of infection for the intestinal protozoa with which the British troops have become and are becoming infected.

(10) Lamblia and trichomonas have been found to occur in Alexandria cats. These animals, with rats and mice, may act as reservoirs of infection.

(11) The introduction of carriers of *E. histolytica* into England at the present time as a result of the movements of troops is unlikely even under existing conditions to be followed by severe outbreaks of amœbic dysentery. In the past carriers have been constantly returning to England without any severe outbreaks resulting.

(12) It is impracticable to examine large bodies of healthy troops with a view to eliminating the carriers of *E. histolytica*. The majority of the carriers are perfectly healthy and we know nothing of the percentage of these carriers which actually pass on to a condition of amœbic dysentery. Nor do we know to what extent a carrier is likely to hand on his infection to another. It is clear that there are many healthy carriers of *E. histolytica* for every one who actually gets amœbic dysentery.

(13) In the case of men invalided for intestinal disorders and are still ill, if *E. histolytica* is found to be present, the condition can be treated, and if clinical recovery takes place this can be followed by discharge to duty. When every able-bodied man is needed for service in time of war it is not reasonable to detain any one just because he happens to be a carrier of *E. histolytica*. The number of undetected carriers amongst the healthy troops is far greater at the present time than amongst those invalided for one cause or another. It is unnecessary to examine all recovered

dysentery convalescents with a view to detecting the carriers of *E. histolytica* or other protozoa.

(14) If, however, it be decided to identify by microscopic examination and to treat every healthy *E. histolytica* carrier amongst any group of convalescents or healthy men it is well to remember that after treatment it is unnecessary to keep all the cases under microscopic control in order to see if relapse will occur, for the results we have obtained show that the carrier is almost certainly cured by a proper course of emetin unless there has been a history of repeated attacks of dysentery.

(15) Similarly men with flagellate infections unless clinically ill should not be invalided from service.

(16) As a result of inquiries we have made and the examination of men who have returned from Gallipoli it appears to us that amœbic dysentery was not as common on the Peninsula during the summer months of 1915 as has sometimes been supposed.

PART II.*

The Characters and Diagnosis of the various Intestinal Protozoa of Man in Egypt with a Description of Three New Forms.

This section of the report has to do mainly with the morphology of the human intestinal Protozoa. We have not attempted to give a complete description of these. This has already been done fairly completely in other papers, but we have noted some important new points and attention is called to these in the various sections below. Finally, we have described three new parasites—two flagellates and an amœba—which have not been previously found in the human intestine.

(1) *Characters and Diagnosis of Unencysted Entamœba histolytica.*

As regards the morphology of this amœba we have very little to add to what has already been so often described. We are convinced that its identification, apart from the presence of included red blood corpuscles, presents the greatest difficulties even for the expert and trained observer. It is true that a certain type of amœba with refractile ectoplasm, indistinct nucleus and active movement is most likely *E. histolytica*, but very often the amœbæ take on quite other appearances and become practically indistinguishable from certain forms of *E. coli*. They sometimes have perfectly distinct nuclei, they may be very sluggish in their movements, they may show little or no distinction between ectoplasm and endoplasm, and they may vary very greatly in the degree of their vacuolation and refractibility. Furthermore, we have seen amœbæ which are undoubtedly *E. coli* moving with an activity which is comparable only with that of *E. histolytica*. Still, as a rule, an amœba is *E. histolytica* if it is moving with an active streaming motion and throwing out pseudopodia, sometimes after several minutes of perfect quiescence, with that peculiarly explosive suddenness which cannot be appreciated unless seen. No amount of description, as James points out, can give an accurate mental picture of this remarkable amœboid activity. It is probably

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* Reprinted from the *Journal of the Royal Army Medical Corps*, February—March, 1917.

true that a certain type of nucleus is more commonly found in *E. histolytica* and another type in *E. coli*, and that in one it is more often visible in the living amœba than in the other, but here again it is exceedingly doubtful if such details of structure can be employed as a basis of diagnosis. Size is of no value whatever in the differentiation of unencysted forms of *E. coli* and *E. histolytica*. It is a very easy matter to state that a certain type of nucleus belongs to *E. histolytica* and another type to *E. coli*, and dogmatically to diagnose amœbæ accordingly, but is there sufficient evidence that the nuclei maintain their characters so consistently as to justify one in making dogmatic assertions on this basis and to condemn patients to courses of emetin and other treatment accordingly?

In examining stools one often sees highly refractile, distinctly green amœbæ with or without vacuolation. As a rule no nucleus can be seen in them and there is either no movement at all or very sluggish change of shape. They may be perfectly round and it may be difficult on account of the refractile edge to be sure that the amœbæ are not encysted. In other cases there may be numerous short conical elevations or irregularities over the whole surface which are hardly pseudopodia and which remind one most of the short pseudopodia of *Amœba verrucosa;* while at other times there may be several rather long, lobose, sometimes branched pseudopodia which move very sluggishly. Amœbæ of this type are met with both in undoubtedly pure *E. coli* infections and in the "carrier" stage of *E. histolytica* infections. It is not only quite impossible to recognize whether these are *E. coli* or *E. histolytica*, but it is often exceedingly difficult to tell whether they are amœbæ at all, especially when they occur in association with the large cells so often seen in the bacillary dysentery exudate. Again, *E. histolytica* in acute cases may be very difficult to identify. It often, of course, has the characteristic structure of distinct ectoplasm and granular or vacuolated endoplasm, but at other times it may be nothing more than a clear hyaline apparently structureless mass, or a mass of such hyaline material filled with numerous vacuoles. We can recollect, having watched a clear hyaline body for some time, wondering the while whether it was a tissue cell on the way to degeneration or an amœba, when suddenly there burst from its side a large pseudopodium and the amœba commenced that series of active movements which one practically never sees except in the case of *E. histolytica*. This particular case had also amœbæ with included red blood corpuscles. We mention these points in order

to show how impossible it is at times to arrive at a diagnosis of the amœbæ themselves and how important it is to take into consideration other features of the cases in which they occur.

Realizing the practical difficulties standing in the way of accurate diagnosis we have fixed a very definite standard for our present series of observations—a standard which one of us has upheld and taught for a considerable time in connection with this work and one which the ordinary observer, who knows how to recognize amœbæ and cysts, can readily follow. We have called no infection one of *E. histolytica* unless we have found at least some amœbæ with included red blood corpuscles present, or unless we could find definite cysts of *E. histolytica* associated with the amœbæ in the stool. It has happened on many occasions that amœbæ have been found in dysenteric and diarrhœic stools which may or may not have been *E. histolytica*, but unless some of the amœbæ contained red blood corpuscles, or unless encysted forms were present, we have left the diagnosis at "free entamœbæ" alone and have followed the cases for several days after the preliminary examinations. Such cases watched from day to day in most instances show cysts of *E. coli* alone in the stool as the symptoms subside, while in a smaller percentage of cases *E. histolytica* cysts appear. In practically all cases in which amœbæ of doubtful nature occur an observation extending over a few days will clear up the diagnosis as cysts make their appearance. There are, however, very rare exceptions to this rule of the appearance of cysts, as has been noted by James in certain cases in Panama. This observer followed three cases of untreated amœbic dysentery for three weeks and no tendency to cyst formation occurred. One of our cases, however, has been watched for over three months, the stool being examined practically every day, and though the case is undoubtedly one of amœbic dysentery as active amœbæ containing red blood corpuscles have been present from time to time during typical attacks of dysentery, on no occasion have cysts of *E. histolytica* appeared. This case has been treated with emetin on several occasions but has always relapsed sooner or later with a return of the dysenteric symptoms. As a rule, however, cysts occur at some time in the course of infections. For instance, in the case of Russell, H., who was admitted with amœbic dysentery, there was blood and mucus in the stool with active amœbæ containing red blood corpuscles. The case was given a twelve-day course of emetin (one and a half grains a day by the mouth and injection) and the symptoms and infection cleared up. A week later cysts of

E. histolytica appeared in the stool, and later still the patient was readmitted to hospital with amœbic dysentery. Of course, in this case the diagnosis was made on the occurrence of red blood corpuscles in the amœbæ, and the correctness of this was proved by the subsequent relapse with the passage of *E. histolytica* cysts.

The greatest difficulty is likely to occur when persons infected with *E. coli* or carriers of *E. histolytica* suffer from bacillary or other forms of dysentery. The encysted amœbæ are not generally present unless the case is seen very early and it may at times be impossible to diagnose accurately the amœbæ, though the absence of included red blood corpuscles is a very strong argument in favour of their being *E. coli*, for infections with this amœbæ are so much commoner in healthy or apparently healthy men. The question is, are all such cases to be treated as if they were amœbic dysentery? If so, we are neglecting the possible bacillary element, so that logically such cases would have to be treated as mixed infections and given both emetin and serum or other bacillary dysentery treatment. One may have to adopt this course in certain cases but a guide to treatment can be obtained in other directions. In the first place, the case may be clinically bacillary rather than amœbic dysentery, and though amœbæ are present, i.e., amœbæ without included red blood corpuscles and unassociated with cysts of *E. histolytica*, it should be treated as bacillary dysentery, for as the symptoms subside cysts of the amœbæ will almost certainly appear and the species be identified. Again, much can be gathered from the character of the stool, and it cannot be too strongly emphasized that it is the duty of every medical officer who has charge of dysentery cases to make arrangements whereby he can see the entire stool of his cases. It is not sufficient either for him or for the person entrusted with the microscopical examination to rely on the patient's statement or to be content with the examination of only a small sample. In many cases this may be sufficient, but, as will be explained below, the picture of the entire stool is so characteristic that a diagnosis can often be made at a glance. Again, the microscopic appearance of the stool apart from the amœbæ is of considerable help. In the examination of a dysenteric stool it is important to examine both the fæcal and mucus parts if both are present, for amœbæ alone may occur in the latter and cysts with or without amœbæ in the former, though this condition of affairs exists only when a carrier case is relapsing into one of acute amœbic dysentery. Case Ball is of interest in this connection ; he was on the staff of the hospital and reported sick with the

passage of blood and mucus. The first stool examined consisted of two parts, a fæcal part and a patch of dark blood-stained mucus. The former contained cysts of *E. histolytica* and free amœbæ of the minuta type in large numbers, while the mucus contained many large active amœbæ, some of which had ingested red blood corpuscles. The patient was evidently a carrier of *E. histolytica* relapsing into an attack of acute dysentery. It must also be remembered that any *E. histolytica* carrier case may suffer from bacillary dysentery though it is very doubtful if in such cases the amœbæ which would be of the minuta form would contain red blood corpuscles. Cysts would only be found at the beginning of such an attack when some fæcal matter was still present and probably the flushing action of the dysenteric process would get rid of most of the free amœbæ as well.

Case Morgan affords an illustration of a case of this type. After only fourteen days in Egypt the patient was taken acutely ill with dysenteric symptoms. There was some fever and the clinical picture was that of bacillary dysentery. Examination of the stool showed the characteristic macroscopic appearance of the disease while microscopically the abundance of pus, mononuclear and macrophage cells, intermingled with red blood corpuscles, further supported this view. In addition, however, the stool contained free amœbæ, none of which included red blood corpuscles, and a fair number of cysts of *E. histolytica*. The amœbic infection had probably been contracted in England, where the patient had served for seven months as orderly in a dysentery hospital. The point of interest is the cause of the attack of dysentery. Unfortunately, no dysentery bacilli were isolated from the stool, but the clinical and other features of the case leave little room for doubt that it was actually one of this disease which was the prevailing type of dysentery at that time. The microscopic appearance of the stool corresponded with this view and the fact that the amœbæ themselves did not contain red blood corpuscles and the presence of cysts indicated that the amœbic infection was not of acute amœbic dysentery type. We, therefore, feel justified in describing the case as one of bacillary dysentery in a man who was acting as a carrier of *E. histolytica*.

Another case of this type deserves mention. The patient (Gundry) was admitted for dysentery and the microscopic examination of the stool showed the characteristic exudate of bacillary dysentery. The case was reported as probably one of bacillary dysentery in spite of the fact that fairly active amœbæ, none of

which contained red blood corpuscles, were present. The case was treated accordingly and the diagnosis was later confirmed bacteriologically by the isolation of a bacillus of the Flexner type. Five days later the patient was passing brown unformed motions free from mucus and some cysts of *E. coli* were found. Free amœbæ had been present every day before this. The following day there were present both cysts of *E. coli* and cysts of *E. histolytica*, and as the patient improved in health these became more numerous, while a large trichomonas infection also appeared. It seems quite clear that this was a case of bacillary dysentery in a man who was a carrier of *E. histolytica*, *E. coli* and trichomonas. The fact that the amœbic infection, which was evidently playing no part in the acute symptoms, was left untreated, did not affect the recovery of the patient from his attack of bacillary dysentery. The *E. histolytica* infection was treated later.

As a result of observations on a long series of cases we would lay down the following rules as a guide to the diagnosis of amœbæ in the stool :—

(*a*) If amœbæ containing red blood corpuscles are present in a stool, whether evidently dysenteric or not, they are *E. histolytica*, and mean that some active dysenteric process is going on.

(*b*) If the actual amœbic dysenteric process is so acute as to demand emetin treatment, then amœbæ with included red cells will almost certainly be present in the stool.

(*c*) If amœbæ, none of which contain red blood corpuscles, are present in a dysenteric stool, then the case is either (*a*) bacillary dysentery (or other form of dysentery) with an infection of *E. coli*, or (*b*) bacillary dysentery (or other form of dysentery) occurring in a carrier case of *E. histolytica* in which there is no active amœbic dysenteric process in progress.

(*d*) In either case mentioned under (*c*) no anti-amœbic treatment is urgent, so that the case can be watched for a few days, during which encysted forms of either *E. coli* or *E. histolytica* will almost certainly appear as the acute symptoms subside, and the diagnosis will be established.

(*e*) Amœbæ, none of which contain red blood corpuscles, and which occur in non-dysenteric stools, may be either *E. coli* or *E. histolytica*. In such cases cysts are nearly always associated with the amœbæ, but if not, treatment for the amœbic infection being never urgent, or not required at all, a diagnosis can be made by examining for a few days till cysts appear, as they invariably do.

It sometimes, though rarely, happens that amœbæ cannot be found in the stool, even after several examinations, when actual amœbic ulceration of the large intestine is present. Amœbic abscess of the liver is not infrequent when no amœbæ can be found in the stool. We have discussed the possibility of bacillary dysentery attacking a person who is infected with *E. coli* or is a carrier of *E. histolytica*; but there is another class of case which needs consideration, though we have not come across an example. These are cases in which the *E. histolytica* is actively concerned in the dysenteric process while a true bacillary dysentery exists also. These would be quite different from cases of bacillary dysentery occurring in carriers in which the *E. histolytica* are not actively concerned. If the case is a carrier of *E. histolytica* and develops bacillary dysentery, the disease which demands urgent treatment is the bacillary infection, but if the case is one of true amœbic dysentery combined with true bacillary dysentery it is probable that both diseases should be treated at once. The diagnosis of such cases can only be made by recognizing the clinical and microscopical appearances of bacillary dysentery, isolating one or more of the dysentery bacilli, and at the same time recognizing in the stool the actively motile amœbæ with their included red blood corpuscles. Such doubly acute cases are naturally of rare occurrence, but they must not be confused with cases of bacillary dysentery in which free forms of *E. coli* or "minuta" forms of *E. histolytica* are present in the stool.

(2) *Characters and Diagnosis of Cysts of E. histolytica.*

The characters of the cysts of *E. histolytica* with the one, two, or four nuclei, the vacuoles and chromidial bodies are now too well known to need further description from us. We would point out, however, how closely the cysts may at times be simulated by the I-cysts, especially, as sometimes happens, these are devoid of I-bodies. In such cases the nucleus may be larger than usual, and it may be exceedingly difficult to decide whether one is dealing with *E. histolytica* cysts or not. This difficulty may be still greater in films containing I-cysts stained by the iron hæmatoxylin method. There the iodophilic body appears not as the refractile structure seen in the unfixed material, but as a vacuolic area by the side of which is the nucleus giving the appearance of a vacuolated *E. histolytica* cyst with a single nucleus (Plate III, figs. 12–17).* In these cases the nucleus is generally smaller than the nucleus of the unenucleated *E. histolytica* cyst, and, moreover, is structurally

* *See inset between pages* 148 *and* 149.

different. The difficulty of diagnosis may be only overcome in some instances by following a case for several days with the careful examination of fresh iodine preparations as a control of the fixed and stained films.

A feature of the *E. histolytica* infection, which the examination of a large series of cases has impressed upon us, is the variation in the size of the cysts. Small forms of *E. histolytica* cysts have been described by James, in Panama, and more recently by Penfold and Woodcock. Kuenen and Swellengrebel described the *E. histolytica* cysts as varying in size between 11 and 19 microns.

We have noticed a similar variation in size, but it appears that various strains of *E. histolytica* occur. There is one which produces very small cysts associated with correspondingly small "minuta" forms of amœbæ. The cysts are 7 to 10 microns in diameter and have the same characters as the larger cysts. These are the forms described by James and by Penfold and Woodcock. The small cysts do not appear to be accidentally small, but cases infected with the small strain pass small cysts regularly, at any rate for some weeks, with no tendency for the small cysts to be replaced by the larger ones. For instance, case Kettlewell was observed for three weeks, during which a twelve-day course of one-grain emetin injection was given without result, and small *E. histolytica* cysts were found constantly. He was finally cured by a course of emetin by the mouth.

Starting from the strain with small cysts, a series of strains occur with gradually increasing average size of cyst. There are strains in which the cysts measure from 9 to 12 microns, others 10 to 14, others 12 to 16, and finally large strains with cysts measuring 14 to 18 microns. As is to be expected, each strain is associated with "minuta" forms of amœbæ of corresponding size.

It seems very improbable that these strains represent different species of amœbæ, for we cannot be sure that a strain of amœbæ which will produce cysts of small size at one time will never at another time produce larger ones. We have noted, however, that in case Healy, in which cysts of large average size were found for a long time, towards the end of the period of observation a certain number of smaller ones began to appear. The point, however, can only be definitely decided by following individual untreated cases for long periods. In order to illustrate this variation we reproduce below a series of measurements in microns made from cysts as they appeared in iodine preparations. Only cysts circular in outline were measured and the cysts were consecutive ones, as they were observed in working through the preparation with the $\frac{1}{12}$-inch objective.

Case Russell, F. (Plate I, figs. 7-9).*—8, 7, 8, 7, 7, 8, 9, 8, 7, 8, 6, 7, 8, 7, 8, 7, 8, 7, 8, 6, 9, 8, 7, 8, 7, 8, 8, 8, 6, 8, 8, 9, 8, 8, 8, 8, 7, 8, 8, 8, 8, 7, 8, 8, 7, 8, 8, 7, 9.

Case Kettlewell.—10, 9, 8, 10, 8, 9, 8, 7, 10, 9, 9, 9, 10, 9, 10, 10, 10, 9, 8, 10, 9, 11, 8, 9, 8, 10, 9, 8, 8, 9, 10, 8, 9, 9, 12, 10, 9, 10, 10, 11, 10, 8, 8, 9, 10, 8, 11, 10, 9, 9.

Case Cooper.—10, 12, 12, 11, 12, 12, 12, 13, 11, 13, 12, 10, 12, 13, 10, 12, 13, 11, 12, 14, 12, 12, 9, 13, 12, 12, 12, 11, 11, 12, 12, 11, 12, 13, 12, 13, 11, 11, 12, 10, 12, 10, 12, 12, 13, 12, 11, 11, 13, 12.

Case Noon.—14, 14, 14, 14, 13, 12, 13, 14, 13, 12, 12, 13, 12, 13, 11, 14, 13, 14, 15, 12, 12, 15, 14, 14, 12, 12, 12, 12, 12, 12, 12, 13, 13, 15, 12, 10, 14, 12, 12, 14.

Case Flynn (Plate I, figs. 4-6).*—14, 12, 12, 12, 13, 12, 12, 12, 14, 14, 13, 14, 14, 12, 12, 11, 13, 14, 14, 12, 14, 14, 12, 11, 13, 12, 13, 14, 12, 14, 13, 12, 15, 14, 10, 12, 14, 12, 14, 12, 14, 12, 14, 14, 15, 13, 11, 12, 14, 13.

Case Healy (Plate I, figs. 1-3).*—16, 18, 14, 14, 12, 14, 15, 18, 14, 16, 16, 14, 18, 18, 18, 14, 16, 16, 16, 15, 16, 15, 14, 15, 14, 16, 14, 16, 18, 18, 16, 15, 16, 12, 14, 14, 15, 12, 12, 13, 17, 14, 14, 16, 16, 14, 12, 15, 16, 14.

The figures (Plate I, figs. 1-9)* represent cysts with one, two, and four nuclei drawn to the same scale from three of the above cases. They show graphically the great difference in size of these various strains. It may, however, be safely stated that the majority of cases of *E. histolytica* infection show cysts with a diameter of 10 to 14 microns, but one must always be careful to recognize the smaller and larger strains, for they have to be carefully distinguished from cysts of *E. nana* on the one hand and cysts of *E. coli* on the other.

The cysts of the large strains differ from the cysts of *E. coli* in the more frequent possession of chromidial bodies and the constant presence of not more than four nuclei.

As regards the virulence of the different strains and their reactions to emetin we can make no very precise statements. Case Kettlewell, who had the small strain, did not clear up on injections of one grain of emetin for twelve days, but did on subsequent treatment by the mouth, and case Healey, who had the largest strain, was the most resistant case we had had, and withstood not only emetin injections, but also emetin and pulv. ipecac. by the mouth.

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* *See inset between pages* 148 *and* 149.

(3) *Are the four Nuclear Cysts of histolytica type necessarily an Indication of Infection with the Pathogenic Amœba?*

All the cases of *E. histolytica* infections have been diagnosed either by the cysts or the presence of amœbæ with included red blood corpuscles: no amœbæ even in dysenteric stools which were not associated with cysts of *E. histolytica*, or with amœbæ showing definite included red blood corpuscles, were entered as *E. histolytica*. The percentage of carrier cases amongst the supposed healthy men, as we have already pointed out, is fairly high and would be actually still higher if allowance were made for the error entering into the single examination system. This being the case, one naturally wonders why, with so many carriers about, is actual amœbic dysentery such an uncommon disease in Egypt at the present time; and secondly, are the tests one applies for the detection of carriers reliable, and are the cysts of *E. histolytica* as one recognizes them in reality the cysts of the pathogenic amœba which produces amœbic dysentery? Doubts on this score have arisen in the minds of some, naturally enough when one realizes the great discrepancy between the number of carriers and acute cases. That the so-called cysts of *E. histolytica* are what they are supposed to be can hardly be doubted by anyone who has followed the literature dealing with the subject during the last few years. Cases of amœbic dysentery have been followed by several observers through convalescence and as the dysenteric symptoms abate the characteristic cysts begin to appear in the stools. Other cases which have been in the "carrier" condition passing cysts in the stools have relapsed into attacks of acute amœbic dysentery. Cysts from carrier cases have been administered to cats and these have contracted acute amœbic dysentery and even amœbic abscess of the liver. Further, Darling has shown that cats infected in this way if they tend to recover from the attack of amœbic dysentery commence passing *E. histolytica* cysts in the stool. The evidence seems to be as complete as it possibly can be, short of some method of maintaining *E. histolytica* in pure artificial culture. Some further evidence in this direction has been the outcome of our observations on cases of amœbic dysentery and carriers in Egypt. Two cases—Blair and Russell, H.—were admitted to hospital with amœbic dysentery, there being blood and mucus stools containing active amœbæ with included red blood corpuscles but no cysts. The cases were treated with emetin and the dysenteric symptoms and the amœbæ disappeared. Both these cases relapsed later. Case Russell, H., began to show cysts of *E. histolytica* with small

amœbæ and a fortnight later had another attack of amœbic dysentery. Case Blair recovered in the same way under emetin treatment, but amœbæ and cysts of *E. histolytica* were present a fortnight after treatment was stopped. Both these cases had had repeated attacks of dysentery before coming under the present observation.

If an individual who is a carrier of *E. histolytica*, and is passing encysted forms in the stool, relapses into acute dysentery one should be able to observe the transition if the case is examined early enough. A case of this kind was seen under very favourable conditions for this examination. A patient (case Ball) was one of the hospital staff who had had repeated attacks of dysentery and much emetin treatment. He was suddenly taken ill with dysentery and the stool was examined almost at once. It consisted of two parts, a fæcal portion and a blood and mucus portion. The former contained numerous cysts of *E. histolytica* with some amœbæ, and the latter numerous active amœbæ with included red blood corpuscles. It was evident that this was an instance of a carrier relapsing into a condition of acute amœbic dysentery. He was treated with emetin and the symptoms and infection vanished, but ten days later cysts of *E. histolytica* were again present. Another course of emetin was given and the infection again disappeared, only to return with cysts and amœbæ three weeks later. In two other cases (Rushforth and Dorter) a similar condition of affairs existed at the first examination, there being acute dysentery with active amœbæ containing red blood corpuscles and associated with cysts of *E. histolytica*. Both these cases relapsed after emetin, and cysts of *E. histolytica* appeared in the stools. In the case of Dorter there was a previous history of much dysentery, whereas with Rushforth there had been no previous dysentery, the patient having only been in the country a short time.

In the majority of cases of actual amœbic dysentery cysts of *E. histolytica* cannot be discovered, though an examination of a fæcal portion of the stool, if such be present, may reveal them when only free amœbæ are to be found in the blood and mucus part. Cases like those recorded above leave little room for doubt that the cysts of *E. histolytica* are definitely related to and derived from the amœbæ which give rise to the dysenteric symptoms. One case, however, must be mentioned, for in it no cysts of *E. histolytica* could be found at any time, though there were repeated attacks of dysentery which were cured temporarily by emetin treatment. This case, Smith, was followed most carefully from April 5 to

July 15, specimens being examined on eighty-eight days. The case was admitted for dysentery, the stool containing blood and mucus and active amœbæ with included red blood corpuscles. Emetin treatment caused the amœbæ and symptoms to disappear but relapse occurred, and at this time there were no cysts but only the amœbæ as on the previous occasion. Another course of emetin was followed by a similar relapse, while a further course of methyl emetin sulphate had no effect on the infection. As already remarked, on no occasion were cysts of *E. histolytica* found, though it must be noted that cysts of *E. coli* were present in small numbers on one or two occasions during the first month of observation —a fact which might appeal to those who wish to claim a pathogenicity for *E. coli*. Why then did not the *E. histolytica* in this case produce any cysts? The only explanation seems to be that the case was in a constant state of acute dysentery. The stool was practically always either liquid or unformed, without any administration of salines, and there was nearly always mucus and often blood mixed with the fæcal matter or separate from it. It is interesting to note that a tetramitus infection which was also present on no occasion produced any cysts, and it may be that the unknown factor which prevented the encystment of the *E. histolytica* prevented also the encystment of the tetramitus. In this particular case, however, the diagnosis was never in doubt, as amœbæ with included red blood corpuscles were frequently found.

It has already been mentioned that several observers have produced dysentery in cats by giving them cysts of *E. histolytica* by the mouth. We have recently repeated this observation with material from case Carr. There was a very large infection of *E. histolytica* cysts—in fact the largest infection we have come across. There was no history of dysentery whatever, so that the case can be looked upon as a healthy carrier. A small quantity of the stool of this case emulsified in saline was poured into the mouth of a kitten, which was then caged with another of the same age. Both kittens became ill with dysentery and died in a week. In both cases there were numerous amœbæ in the large intestine and both showed extensive ulceration. The experiment is valuable in showing that the cysts of *E. histolytica* in a perfectly healthy carrier, who had no history of dysentery, and who was only discovered to be a carrier in the routine examination of healthy men in camp, were capable of giving rise to a fatal dysentery in animals. Furthermore, it is evident that the second kitten contracted infection from the first, probably just after feeding by licking material

which was adhering to the fur about the mouth of the first. An experiment of this nature suggests very strongly that the cysts from such a carrier case might give rise to acute dysentery in another individual and that the same individual himself might pass into a condition of acute dysentery at a later date. A reference to the protocols will show that case Carr was cured by a course of emetin.

The arguments in favour of the cysts of *E. histolytica* being actually the cysts of the pathogenic entamœba are therefore these :—

(1) Cases which are passing cysts of *E. histolytica* may relapse into acute dysentery when amœbæ with included red blood corpuscles appear in the stool.

(2) Cases of acute amœbic dysentery showing amœbæ with included red blood corpuscles may recover naturally and in the process the large active amœbæ become replaced by smaller forms and cysts of *E. histolytica.*

(3) Cases of acute amœbic dysentery of the above type may be cured with emetin, but often such cases relapse when cysts of *E. histolytica* appear in the stool.

(4) Cysts from perfectly healthy carrier cases who have no previous history of dysentery will give rise to fatal amœbic dysentery in kittens.

Taking all these points into consideration, there can be no doubt that the detection of the characteristic cysts of *E. histolytica* in the stool is in reality an indication that infection with the pathogenic amœba exists.

(4) *The History of Carriers of E. histolytica.*

Having decided that the cysts of *E. histolytica* are actually the cysts of the pathogenic amœba, it becomes of the utmost importance to know what happens to such carrier cases, how many of them actually suffer from dysentery at some time or other, how many of them recover spontaneously, how long they may remain carriers and what is the condition of the large intestine while they carry their infections.

(*a*) *How long do Carriers remain infected ?*—This is a question which only very prolonged observations can answer. In certain cases, however, long histories of repeated attacks of dysentery can be obtained. For instance, case Healy, who proved so resistant to emetin treatment, had suffered off and on for five years and when examined he was just recovering from an acute attack of dysentery, and was again passing into a condition of a carrier with cysts and

amœbæ in the stool. Another case, Spiers, gave a similar history of a slightly shorter duration. Another case of this kind was seen by one of us (C. M. W.) in London some years ago, and was one of a postman who had been invalided from the West Indies on account of dysentery. He stated he had had repeated attacks during a period of five years since his return to England. Examination of the stool showed that it was soft and unformed and was dotted over with small flakes and streaks of mucus as is common in these cases. There was a very large infection of cysts of *E. histolytica* and small amœbæ.

These cases show clearly that individuals may remain as unhealthy carriers over long periods and as far as we can judge it may be for the rest of life.

The length of time that a person may remain a healthy carrier is much more difficult to decide, for there is no history of repeated dysentery to guide us. One case of known infection has been under observation for six months, and though cysts of *E. histolytica* have been present constantly there has been no dysentery during this period, nor was there a previous history of dysentery or diarrhœa. This case (Cox) was given a course of emetin injections, which only caused the cysts to disappear from his stool for a short time.*

That infections of this kind can exist for long periods without any symptoms is borne out by the fact that such a large percentage of the "carriers" we have found during the routine examination of healthy men have given no history of dysentery whatever. For instance, of 106 carriers discovered amongst 1,979 healthy men only 16 gave any history of dysentery, and it is certain that the latter figure is too high, for in no case can we be certain of the type of dysentery from which the case suffered.

In a certain number of instances men who have been found to be carriers have been brought into hospital for treatment, and while under control before treatment was commenced it was noticed that the infection began to decline and finally disappeared. Three such cases were examined regularly for fifteen, eighteen and twenty-six days respectively after the spontaneous disappearance of the *E. histolytica* cysts from the stool without there being any recurrence. It was impossible to retain these men any longer, but whether the observation indicates a spontaneous recovery or

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* After the lapse of a further twelve months Mr. R. E. Savage has kindly re-examined the case. There has been no dysentery while *E. histolytica* is still present. This case has, therefore, remained neglected for at least one and a half years without symptoms attributable to the infection.

not, it shows that an infection may disappear for a considerable period. In none of these cases was the infection a large one. On the other hand, in the great majority of our cases which were controlled for a week or ten days before treatment was commenced there was no tendency for the infection to disappear as in such cases as that of Healy who was controlled for nearly three months and was rarely free from cysts of *E. histolytica*.

(*b*) *What Percentage of Carriers pass on to an Acute Dysenteric Condition?*—This question is even more difficult than the preceding one, for an answer can only be obtained by controlling cases for long periods and this has not yet been done to any extent. It is clear that many cases can carry their infections for, at any rate, many months without symptoms and it is equally clear that others may have repeated attacks of dysentery in a similar period. Between these two extremes there are many intermediate types where infected individuals have mild attacks of diarrhœa with or without mucus in the stool, which may or may not be the result of the *E. histolytica* infection. A certain number of carriers complain of pain over the large intestine or of other symptoms which are difficult to explain except on the assumption that some intestinal ulcers exist. Others say that they never pass a really formed stool. It is perfectly clear, however, that a large percentage of men who are stationed even for a short time in Egypt become infected with *E. histolytica*. A certain though yet unknown, but by no means negligible, percentage of these pass on to a condition of amœbic dysentery and have to be invalided from the Service. It is evident, therefore, that in the transference of new troops from England to non-infected centres, it is inadvisable to station them in the first place in endemic centres of amœbic dysentery like Egypt unless important military requirements leave no other alternative.

(*c*) *What is the Condition of the Large Intestine in the Carrier?*—It has been suggested that *E. histolytica* can live in the large intestine as *E. coli* does without producing any lesion of the gut, and that this is the condition of affairs in the carrier cases as distinguished from the amœbic dysenterics who have definite ulcerations. It seems, however, very doubtful if this is actually the case, for many facts seem to indicate that some ulceration of the intestine exists in these cases, though it produces no symptoms.

It is a well-known fact that at *post-mortem* examinations in countries where amœbic dysentery is endemic, amœbic ulceration of the large intestine is often encountered in cases in which there is absolutely no history of previous dysentery. In 1910

Musgrave published an account of fifty such cases in the Philippines. Others have had a similar experience elsewhere, and quite recently the Thompsons (*Journal of the Royal Army Medical Corps*, June, 1916) state that Bartlett had noted amœbic ulceration in the intestine of soldiers from Gallipoli who had died of wounds or other cause when amœbic dysentery was not suspected.

It is a remarkable fact that ulceration of the large intestine, sometimes quite extensive, can exist without giving rise to symptoms. It is probable that in these cases where amœbic ulceration was found after death, though quite unsuspected during life, an examination of the stool would have revealed the condition of the carrier case with cysts and free amœbæ in the stool.

In cases, such as those of Healy and Spiers mentioned above, where there is a long history of repeated attacks of dysentery extending over several years, and where during the intervals between the attacks the stools are never normal, always being soft and mixed with a certain amount of mucus, it is impossible to doubt that there exists an extensive ulceration of the intestine. In fact, thickening of the large intestine and painful areas can be found on palpation. These cases during the interval between attacks of dysentery show only cysts of *E. histolytica* and small amœbæ generally in very large numbers. In other cases between the dysenteric attacks the stool may approach the normal and mucus be not apparent, though the number of cysts and amœbæ passed may be very great. Furthermore, there are many carriers who give a history of one attack of dysentery. In them there must have been ulceration at this time, and it seems probable that such ulceration must persist when acute symptoms are in abeyance. Finally, there are the carriers who give no history of dysentery whatever. In many of these a careful examination of the stool will often reveal small flakes and streaks of mucus, and though its presence is not necessarily an indication of acute ulceration, it is proof that some abnormal condition of the intestine exists, for it must be remembered that in the worst and most persistent cases with undoubted ulceration present the condition of the stool between the actual attacks of dysentery may only be abnormal in that it is usually soft and unformed with a certain amount of mucus present. In fact, the stool in these intervals may be both macro- and microscopically exactly like the stool of a carrier who gives no history of dysentery whatever.

It is to be noted that a small percentage of these healthy carriers complain of pain over the large intestine.

The development of liver abscess in cases which give no history of dysentery is far from uncommon, and it seems justifiable to assume that these cases must have been carriers of *E. histolytica* and must have suffered from unrecognized ulceration of the large intestine.

Taking all these points into consideration it seems safe to assume that the *E. histolytica* in the intestine of the carrier case is not comparable with the perfectly harmless *E. coli*, but that it is the cause of a certain amount of ulceration, and we know that this ulceration may be quite extensive without giving rise to any definite dysenteric symptoms.

Another argument in favour of this view is the remarkable action of emetin on the *E. histolytica* infections and its comparative inaction on those of *E. coli*. It seems difficult to understand why the drug should be more toxic to one than another amœba if they were both living under similar conditions in the large intestine. Yet administered by injection to a healthy carrier showing infections of both *E. histolytica* and *E. coli*, it causes the former to disappear, while the latter, as a rule, persists. It seems most probable that the drug given in this manner reaches the *E. histolytica* more easily than the *E. coli*, and the only way in which this could occur would be if the *E. histolytica* had a different habitat from the *E. coli*. This difference can be readily explained if we assume that, in the carrier, the *E. histolytica* is living in or about intestinal ulcers while the *E. coli* is more uniformly distributed over the healthy gut. The injected drug would thus reach the *E. histolytica* through the circulation, while the *E. coli* would escape. This view is supported by the fact that emetin administered by the mouth generally causes temporary disappearance of both *E. histolytica* and *E. coli*. Though we assume that infection with *E. histolytica* means ulceration of the large intestine it still remains a fact that the type of ulceration in a carrier case must be different from that in an acute dysenteric. The ulcer in the carrier is probably of a more indolent nature without there being active destruction of tissue or outpouring of exudate from its surface. Living in the deeper tissues of the ulcer are large amœbæ, while towards the surface of the ulcer, and possibly to a large extent over the surface of the gut around the ulcer, are smaller amœbæ which have been produced by successive divisions of the larger forms. Here on the surface these small amœbæ, the minuta forms, become encysted and produce the typical cysts of *E. histolytica*, which escape in the stool. If, for some reason, such an indolent ulcer becomes acute

there is a greater activity and multiplication on the part of the deep-seated large amœbæ and a greater outpouring of exudate with blood and mucus. In this process the small amœbæ and cysts on the surface of the ulcer are washed away and will only be found in the stool at the commencement of the dysenteric attack, while the now active large amœbæ escape from the ulcer in the exudate, and appear in the blood and mucus stool where they are seen actively crawling about with included red blood corpuscles. When the dysentery abates the ulcer returns to its more indolent state, while the small amœbæ are again produced and the cysts reappear. An acute attack of dysentery may arise, however, not from an increase in the activity of an existing indolent ulcer, but by a fresh attack on some still healthy part where a new ulceration is being established.

The above conception, admittedly somewhat theoretical, fits in well with what we know of the history of many of the carrier cases of *E. histolytica*, and affords a possible explanation of the difference in the action of emetin on *E. histolytica* and *E. coli*.

(*d*) *How does E. histolytica establish itself in the Human Intestine?*—Individuals become infected with *E. histolytica* by ingesting the well-known four nuclear cysts. This has been experimentally proved in cats by many observers, and by Walker and Sellards (1912-1913) in the case of human beings. As there occur so many carriers who have never suffered from dysentery it is clear that many become infected with *E. histolytica* without showing any signs of their infection, at any rate, for some time. In their experimental infection of human beings Walker and Sellards found that only a small percentage (four out of eighteen) of those who became infected actually developed definite dysenteric symptoms. It would seem probable that in nature too the majority of those who become infected acquire at first a benign infection which can only be recognized by an examination of the stool, and that it is only later that the ulcerative process becomes acute or extensive enough to give rise to dysenteric symptoms. In other words, the cases become first of all healthy carriers and then only later lapse into a condition of amœbic dysentery. It is but rarely that one has an opportunity of obtaining evidence to support this view. Case Rushforth, already referred to above, was seen at the commencement of his first attack of dysentery, and in his case cysts of *E. histolytica* were found as well as active amœbæ with included red blood corpuscles. It would seem that the case had been a carrier of *E. histolytica* without showing any symptoms, and was just then lapsing into a

condition of acute amœbic dysentery. Though this is probably the commonest mode of onset of amœbic dysentery, in a certain number of cases actual dysentery sets in soon after infection has taken place without there having been a previous "carrier" period. This was the experience of Walker and Sellards in their infection experiments, where a small percentage of the men who became infected had attacks of amœbic dysentery within a comparatively short time (ten days or more) of ingesting the infective material. In cats, again, it is usual for actual dysentery to occur without any intervening "carrier" period; in fact, no one, as far as we are aware, has yet produced a "carrier" condition in a cat without there having been actual dysentery first. These primarily acute cases, as with the cases of dysentery which are lapses from a "carrier" condition, naturally clear up clinically without treatment, and pass into the carrier condition, which must be looked upon as the most usual normal type of infection with *E. histolytica.*

The idea that a parasite should give rise in most cases to very few or no symptoms at all in its host is not strange in any way and is merely an indication of an adaptation of host and parasite to each other, a condition of affairs which is, so to speak, the ideal arrangement aimed at by a parasite. Any parasite which quickly destroys its host is very soon likely to be destroyed itself.

E. histolytica infections may therefore be established in the following ways after ingestion of the infective cysts:—

(1) The case may become a carrier case showing amœbæ and cysts in the stool without there having been any evidence of previous dysentery and without any tendency to the development of acute symptoms.

(2) The case may have a primary attack of amœbic dysentery and pass on into the carrier condition later.

(3) The case may become a carrier and then lapse into a condition of amœbic dysentery only to become a carrier again as the symptoms subside.

As already mentioned above it is possible that the great majority of carriers eventually show dysenteric symptoms, but as yet we have a very few data to go upon. Actual amœbic dysentery is essentially a chronic disease and persons who suffer from it are constantly having attacks of dysentery alternating with periods of freedom from the acute symptoms. During the attacks amœbæ with included red blood cells can be found in the stool while between the attacks there occur cysts of *E. histolytica* and small minuta amœbæ. During the attacks the stools contain blood and

mucus, while between the attacks the stools may approach the normal or, what is more usual, they remain soft and mucoid, being of a peculiar sticky character, due to admixture with mucus in the form of small flakes or streaks.

(5) *Character and Diagnosis of the Dysenteric Stool.*

It has already been stated that much information as to the type of dysentery present can be gathered from the features of the stool. The typical bacillary dysentery stool is so characteristic that with very little experience one can recognize the condition at a glance. The amœbic dysentery stool is also fairly typical but not so much so as that of bacillary dysentery.

(*a*) *The Character of the Stool in Bacillary Dysentery.*— Bacillary dysentery sets in with acute diarrhœa, which very soon washes all the fæcal matter from the gut, and it is after this that there is found the glairy white or yellowish white mucus streaked or flecked with blood which is very little altered in colour. When seen in the bed-pan this mucus may be sufficiently liquid to pour to and fro or it may be more tenacious and adhere to the bottom of the vessel. It may be the only material present, or there may be a certain amount of fæcal matter between patches of mucus. A small portion of this mucus examined under the microscope shows a varying number of red blood corpuscles on a white field composed almost entirely of pus cells, a smaller number of larger round mononuclear cells and a still smaller number of very large cells which remind one of nothing so much as the large hypertrophied endothelial cells of the blood-vessels. This large type of cell is very commonly found in certain diseases and is generally known as the macrophage. In kala-azar it is the large cell which contains the Leishmania and in malaria it may be found in the peripheral blood. It seems to us that the large cells seen in bacillary dysentery probably have a similar origin. Any of the cells described above may be phagocytic, especially the round mononuclear cells and those of the macrophage type. They frequently ingest the polynuclear pus cells and also red blood corpuscles and it is this feature which renders their confusion with amœbæ such an easy matter for the uninitiated. The macrophages, however, are non-motile, or practically so, even in the perfectly fresh stool. Another feature of this overwhelming cellular exudate is that the individual cells may appear fairly healthy or they may show excessive necrosis, being reduced to nothing more than vesicles, the limits of which are the remains of the cytoplasm or nuclear

membrane. The cells show all kinds of granular degenerative changes, but very frequently one finds greenish homogeneous highly refractile spheres in the cytoplasm of the cells. These spheres are probably of a fatty nature, but their importance, from the present point of view, is that they have been mistaken for nuclei of amœbæ or even red blood corpuscles. It is well to remember that the nucleus of an amœba is never a homogeneous refractile body, but is recognized by the ring of granules at its periphery, while the interior appears of much the same colour as the cytoplasm outside. While the cells which have been mentioned above are the ones most commonly seen in the mucus of bacillary dysentery there occur sometimes patches of mucus showing another type of cell. These are elongated cells changed and distorted in various ways and are evidently derived from the columnar cells of the gut wall itself. This type of cell occurs very commonly in the mucus one so often sees around the formed fæces of the post-dysenteric condition. In these cases the cells seem to have originated from mucous membrane of the lower part of the large intestine. The pus and other cells described above are probably purely exudate cells derived from the outpouring of liquid from lymphatics and blood-vessels, while the latter are exfoliations from the gut wall. The two plates are of outline drawings of cells made from a bacillary dysentery stool and illustrate the cellular exudate on the one hand and the exfoliation type of cell on the other (text figs. 1 and 2, *pages* 62 *and* 63). If films of this cellular exudate are stained after wet fixation by iron hæmatoxylin all kinds of strange pictures are developed, which are a result of the active phagocytosis and nuclear degeneration which is taking place. The pus cells with their fragmented nuclei when four portions are present may simulate the cysts of *E. histolytica*. The remains of the nuclei of phagocyted cells in the cytoplasm of larger cells may suggest reproductive phases, such as schizogony of some protozoon, while it may be impossible at times to distinguish between degenerating cells and degenerating amœbæ. With such a collection of curious and unusual objects before one it is necessary to be on one's guard against errors of interpretation. The characteristic features both macro- and microscopic of the bacillary dysentery stool have been insisted upon by observers before this. Bahr, in his account of bacillary dysentery in Fiji, draws attention to the large cells which may be mistaken for amœbæ. Stitt in his "Manual of Tropical Diseases" repeatedly refers to the characters of the bacillary dysentery stool, the type of cell found in the cellular exudate, and

TEXT FIG. 1.—A composite plate showing the different cells met with in the mucus of a bacillary dysentery stool. Actually the pus cells are more numerous than represented in the plate. They form at least ninety per cent. of all the cells present. The small rings and irregularly shaped bodies with clear interiors are red blood corpuscles.

(*See page* 61.)

warns his readers against the error of mistaking some of these cells for amœbæ. Quite recently Willmore and Sherman have again recognized the value of the microscopic appearance of the cellular

TEXT FIG. 2.—Another appearance met with in the mucus of a bacillary dysentery stool. The cells here are evidently the superficial gut cells in various stages of degeneration. They are not nearly so commonly seen as the cells shown in text fig. 1. Cells of this type are often seen in mucus derived from the gut in conditions other than bacillary dysentery. This drawing was actually made from a single microscopic field.

(*See page* 61.)

exudate as an aid to the diagnosis of bacillary dysentery. With this view we entirely agree. So convinced are we of this characteristic appearance that the cases we have examined are returned as

"probably bacillary dysentery" if it is present, and the cases are treated accordingly. From the point of view of the patient this is most important, for bacteriological examination cannot give an answer quickly enough. Theoretically it may be possible to isolate and diagnose the bacillus in thirty-six to forty-eight hours, or occasionally in eighteen hours, but this is the critical period for the patient and it is during this period that active anti-dysenteric treatment should be adopted. As a matter of fact, in practice the bacteriological diagnosis takes longer than forty-eight hours on an average, so that frequently all signs of dysentery have vanished before the report is obtained. It is obvious therefore that bacteriological diagnosis is too slow to be of use in assisting at the early treatment and one must have recourse to other methods—viz., a consideration of the clinical characters of the case, the macroscopic appearance of the stool and the microscopic appearance of the exudate. While the type of stool described above is typical of the bacillary dysenteric attack at one period of its development, it must not be forgotten that before and after this the stools may be very different. Often a patient does not report sick till the blood and mucus appear, and by the time that he is in hospital it may have vanished. The character of the stools after the blood and mucus stage depends largely on the type of diet the patient has had. As a rule he has been too ill to want much food. Often at this stage one sees what we have called the brown liquid stool which differs macroscopically in no way from the stool which would result from a saline purge. Microscopically, however, one may find all the cells present which one finds in the typical bacillary dysentery mucus. They are, however, uniformly distributed through the liquid fæcal matter, though patches of cell agglomerations may occur here and there. This appearance probably indicates a bacillary dysentery which has passed the mucus stage, or a case which will not develop the mucus stage at all. In reporting on these cases, however, much greater caution is needed, for a stool of this kind may be produced by chronic amœbic conditions or by the ulceration of the large intestine which is left after the amœbic infection has disappeared after treatment. A case illustrating this point is described below. At other times in bacillary dysentery one sees a stool like the rice water stool of cholera, and here again there is an abundance of pus, mononuclear cells and large macrophages. As the dysenteric condition passes off the cellular elements diminish till finally the microscope fails to yield any information.

(*b*) *The Character of the Stool in Amœbic Dysentery.*—When we come to the characters of the stool of amœbic dysentery the difficulties to be encountered are much greater. When actual dysentery is present the blood and mucus are much darker in appearance than the blood and mucus of bacillary dysentery. The blood may be black or brown, while the mucus is often transparent and dark brown in colour. The blood and mucus again are more often mixed up with fæcal matter, and one does not have the picture of the bottom of a pan covered with whitish mucus and blood as in bacillary dysentery. Again, in amœbic dysentery the stool may be merely a soft unformed stool, which on close examination is found to be impregnated with mucus intimately mixed with the fæcal matter. Such a stool may be termed mucoid.

Microscopic examination of the stools of the amœbic dysenteric shows nothing characteristic apart from the amœbæ. Cells of many kinds are present, in fact any of the cells described as occurring in bacillary dysentery. But the cells are never present in such numbers and one does not find that condition where the whole field is covered with them. There are then no cells characteristic of amœbic dysentery, but the absence of the bacillary dysentery picture may lead one to assert that the case is probably not bacillary. A diagnosis can only be arrived at by finding the amœbæ with their included red blood cells. If these are not present the case may be one of some other disease, but may still be amœbic dysentery, for in these cases it sometimes happens that the amœbæ cannot be found at the first examination.

Another and very important point which must not be forgotten in these cases is that a negative bacteriological or protozoological examination does not exclude the disease. It is probably true that a microscopic examination of amœbic dysenteric stools will give a positive result in a few minutes more frequently than a bacteriological examination of a bacillary dysentery stool will in as many days; but even the protozoological examination will fail sometimes. To avoid such errors the stools should be examined on several occasions, when the chances of error will be reduced to a minimum.

An instructive case in point and which has been referred to above is the following. The patient, who had been invalided from Mesopotamia with dysentery, was admitted to hospital in Alexandria. The stool, which was a brown liquid one, did not contain evident blood and mucus but microscopically there were present numerous blood, pus and other cells reminding one of the picture of a bacillary dysentery in the post-mucous stage with liquid stool. No amœbæ

were found. The stool was examined on two other occasions with a similar result. The bacteriological examination was also negative. It was assumed to be a case of bacillary dysentery, and as the diarrhœa continued with the passage of blood and pus cells the case was treated with repeated injections of serum. An irregular temperature developed and the patient eventually died. At the post-mortem the upper part of the large intestine showed extensive amœbic ulceration while the lower half was denuded of mucous membrane, save for a few scattered islets here and there. There was also a large abscess of the right lobe of the liver, which formed a mass adherent to lung and diaphragm. Examination of the liver abscess pus showed numerous active *E. histolytica*, while scrapings from the intestinal ulcers showed no amœbæ at all. This case had previously been treated with emetin, so that it is possible the intestinal infection had vanished while the liver infection had remained. The important point of the case is that the microscopic examination of the stool on three occasions failed to give a diagnosis of the true condition, which in this instance could only have been arrived at by clinical methods. The bacteriological and protozoological examination may give a definite and conclusive answer in most cases, but the clinician must remember that he must help in the diagnosis to some extent however empirical his methods may be.

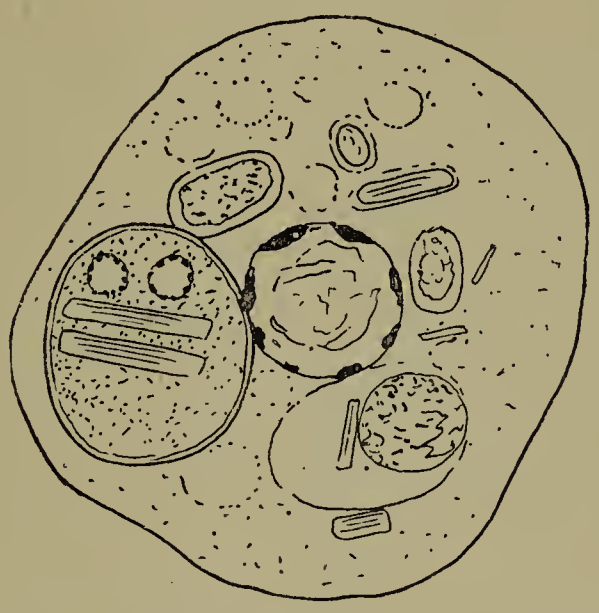
(6) *Characters and Diagnosis of Unencysted E. coli.*

Our remarks as to the difficulty of identifying the free unencysted forms of *E. histolytica* apply equally to *E. coli*. There is a type of amœba which one sees in non-dysenteric cases which can almost certainly be recognized as *E. coli* without the presence of the eight nuclear cysts. These are amœbæ with a thin rim of pale not highly refractile ectoplasm enclosing an endoplasm which is much vacuolated and which contains bacteria, bacilli, yeasts and other objects, while the rather large nucleus, distinct because of the coarse granules of chromatin on its membrane, can be clearly distinguished. These amœbæ move sluggishly as a rule and throw out pale non-refractile pseudopodia. *E. coli*, however, frequently departs from this type, and as already mentioned we have seen *E. coli* moving as actively as any *E. histolytica* does. The character of the amœbæ changes also, so that they may resemble certain forms of *E. histolytica* structurally, and it is possible this change in appearance is dependent in some way upon the character of the stool. *E. coli* in a dysenteric or a diarrhœic stool never has the same appearance as when it occurs in a normal stool.

In an absolute diagnosis of this amœba we must rely on finding the characteristic cysts, for we have no such criterion as the ingestion of red blood corpuscles to guide us.

Amœbæ, however, which occur in a dysenteric stool and which do not, any of them, contain red blood corpuscles are most probably *E. coli* for the simple reason that *E. coli* infections are so much more common in healthy individuals than *E. histolytica* infections.

If amœbæ show many vacuoles, especially vacuoles which are large elongated almost rectangular fissures, they are probably *E. coli*. The remarks made under *E. histolytica* as to the propriety of waiting for a diagnosis when only unencysted amœbæ can be found apply equally here, for no one in these days would advocate



TEXT FIG. 3.—*Entamœba coli* with ingested cyst of *E. histolytica* (two-nuclear stage). Case Boyd, May 13, 1916.

(*See page* 175.)

treating all amœbic infections with emetin. When free amœbæ alone are found, the following of a case daily for a few days will almost certainly reveal encysted forms. In one case only, which had a persistent diarrhœa, free amœbæ alone were passed for nearly a fortnight before *E. coli* cysts appeared.

E. coli is much more omnivorous than *E. histolytica* and one more frequently finds bacilli, cocci, yeasts, long coiled up leptothrix and unidentifiable structures within the cytoplasm. On two occasions we have seen a large amœba, almost certainly *E. coli*, which had phagocyted a cyst of *E. histolytica*. A drawing of one of these is reproduced (text fig. 3, *above*). Cysts of lamblia are also ingested by *E. coli*. Though bacteria of all kinds are taken up readily by *E. coli*, one sometimes finds bacteria within undoubted *E. histolytica*. On several occasions we have noted short bright

refractile rods in *E. histolytica* as well as in *E. coli*. These are spores of a spore-bearing bacillus (probably *B. megatherium*) which is fairly common in fæces in Egypt (text fig. 4, *page* 85). On another occasion most of the amœbæ in an *E. histolytica* infection had ingested larger oval refractile structures which were probably yeasts.

Does E. coli ingest Red Blood Corpuscles? Some observers have declared that *E. coli* may, under certain circumstances, phagocyte red blood corpuscles. They have not, however, told us why the entamœbæ observed could not have been *E. histolytica*. If one decides that a certain type of nucleus or cytoplasm must of necessity belong to *E. coli*, then of course one is bound to declare any amœba, with such a nucleus, whether it contains red blood corpuscles or not, to be *E. coli*. For instance, James, discussing this question, states that in mixed infection of *E. coli* and *E. histolytica* in stools containing blood he has found red blood corpuscles in varying quantity in the interior of the pathogenic organisms but rarely within coincident *E. coli*, and then only one or two at a time. Is it not possible that in such cases the amœbæ were really *E. histolytica* which had come very near to *E. coli* in structure? In order, therefore, to test the powers of *E. coli* to ingest red blood corpuscles we have made some experiments about which there could be no shadow of doubt. A case of pure *E. coli* infection which had been followed continuously for nearly four months was used. On two occasions when the free amœbæ were present in large numbers a portion of the perfectly fresh stool was mixed with a quantity of finger blood of the infected individual on one occasion and with the blood of another individual on a second. The mixture was placed at once in the warm incubator and examined from time to time. The amœbæ were moving freely amongst the blood cells, and though the red blood corpuscles became adherent to the surface of the amœbæ none of them were ingested. It is clear, therefore, that *E. coli* does not readily ingest red blood corpuscles under the conditions of the experiment.

Our observations in cases of bacillary dysentery with much blood in the stool, and where *E. coli* were present as demonstrated by the subsequent occurrence of *E. coli* cysts alone a few days later, all go to show that even in the gut where there is an abundance of red blood corpuscles present the amœbæ do not tend to ingest them. On the contrary, all cases with amœbæ showing red blood corpuscles have proved on careful investigation to be cases of *E. histolytica* infection.

On the other hand, the experiment outside the body we have repeated with the small form of *E. histolytica* which occurs in carrier cases. Though the amœbæ were moving actively they did not ingest the red blood corpuscles, so that it would seem that those amœbæ which one finds in the stool with included red blood corpuscles must be amœbæ which have escaped from some definite active lesion of the gut, where they have been living as tissue parasites. These observations lend support to the view which we have expressed elsewhere, that if amœbæ are found with phagocyted red blood corpuscles they are certainly *E. histolytica*, and are taking part in some active dysenteric process, and that such cases without further evidence require emetin or other anti-amœbic treatment.

Only on one single occasion, as we have noted above, has free *E. histolytica* with included red blood corpuscles been found in an ordinary unformed stool which showed no evidence of blood or mucus either micro- or macroscopically. In this case there were present numbers of rather small amœbæ which were thought to be minuta forms of *E. histolytica*. A search for *E. histolytica* cysts was being made when a large active amœba with several red blood cells within it was found. No cysts were present. The case was diagnosed accordingly and a few days later the diagnosis was confirmed by the finding of numerous cysts of *E. histolytica*.

(7) *Characters and Diagnosis of Cysts of Entamœba coli.*

As regards the cysts of *E. coli* we have very little new information to offer. We have noted, however, greater range in size than has been previously admitted. James, describing *E. coli*, has noted cysts as small as 10 microns in diameter. Undoubted *E. coli* cysts as small as this we have not seen. The lowest limit of size has been 13 or 14 microns.

We have frequently seen infections in which practically all the cysts were over 20 microns, while once the average size was well over 25, and one cyst circular in outline had a diameter of 32 microns ; while on another occasion a cyst measuring 38 by 34 microns was seen. These large cysts often show sixteen instead of the eight nuclei so characteristic of *E. coli*. It is very probable that strains of *E. coli* exist as we have described for *E. histolytica*, one strain differing from another in the average size of its cysts. We have not, however, made any definite measurements to decide this point.

Another feature of the cysts of *E. coli* which demands attention is the possible presence of chromidial bodies. These may be single bars, very like the rods in *E. histolytica* cysts, or they may be multiple when they take on various shapes, being either rod-like, round, oval, or spindle-shaped masses, or more irregular in form. Sometimes in *E. coli* cysts there occur numerous fine filamentous structures which may show a thickening in the centre, so that they are fusiform in shape. These may be arranged irregularly through the cytoplasm with the eight nuclei distributed amongst them, or the eight nuclei may be grouped together in a central mass of cytoplasm, while the fusiform bodies are arranged tangentially around this central mass. The fusiform structures may be few in number, or very numerous. We have some evidence that they are really bacteria which have not been extruded by the encysting amœbæ. The possibility of their being parasites of the amœba has also to be considered.

It has been pointed out before that the type of *E. coli* cyst most commonly met with in the stools is the eight-nuclear cyst, while the stage with two or four nuclei is more rarely seen. An exception to this rule, however, is the frequent occurrence of the type of cyst with large central vacuole and only two nuclei. Sometimes nearly every cyst in a stool is of this type. These bi-nuclear, vacuolated cysts are generally quite green in colour, and more refractile than the clear transparent non-vacuolated eight-nuclear cysts. It seems to us that there is something abnormal about these cysts, and it still remains to be proved that they are capable of further development. We cannot agree with Kuenen and Swellengrebel that these two-nuclear vacuolated cysts are an essential stage in the development towards the unvacuolated eight-nuclear cysts. As a matter of fact, the one, two, and four-nuclear cysts without the central vacuole are far from uncommon when one happens to get a stool containing the actually encysting amœbæ. The two-nuclear cysts with central vacuole seem to be derived from certain green highly refractile amœbæ which have a much vacuolated cytoplasm. They are fairly common, and are often seen as green discs of cytoplasm, the central part of which is either a single large vacuole or several smaller ones. The nucleus in these amœbæ may or may not be clearly visible.

Suggestions have recently been made that the distinction between the cysts of *E. coli* and *E. histolytica* are not so definite and marked as some maintain. It has been even hinted that the one may be only a smaller variety of the other. Gauducheau has

written that the distinctions between *E. coli* and *E. histolytica* will not hold. We quite admit the difficulty of distinguishing vegetative forms of the amœbæ, and recognize that occasionally there may be some uncertainty even with the cysts, for large strains of *E. histolytica* cysts occur as well as small strains of *E. coli*. *E. coli* cysts may have chromidial bodies, but the presence of four nuclei in the fully developed *E. histolytica* cysts is so universal that one can state with certainty that this is a reliable feature for diagnostic purposes. The *E. coli* cysts when developed have eight nuclei, and sometimes more. The clearest demonstration of the difference of the two cysts is seen in cases of mixed infections treated with emetin where the *E. coli* cysts survive the treatment, and the smaller four-nuclear *E. histolytica* cysts disappear; while in pure *E. histolytica* infections there is a complete disappearance of all the cysts. Such cases are absolutely impossible to explain on any other basis than that the two amœbæ, *E. coli* and *E. histolytica*, represent two distinct species of different characters and habits.

(8) *The Course of E. coli Infections.*

It has already been pointed out by one of us (C. M. W.) that *E. coli* infections may persist for several years. James, working in Panama, mentions a case which constantly showed *E. coli* during a period of about five years. A case of *E. coli* infection which was not complicated by the presence of any other protozoon we have followed very carefully during the last few months. The results of the examinations made during a period of 120 days are shown in the accompanying table (*page* 72). The character of the stool at each examination and the occasions on which a saline purge was administered are set down, for it will be seen how directly this affects the finding. Encysted forms were present on most occasions, whereas the free unencysted forms were rarely present except when the stool was soft after the saline purge. On the ninety-fifth and ninety-sixth days there was diarrhœa accompanied by a very large blastocystis infection. This case illustrates very well what may be taken as the normal course of an *E. coli* infection judged by the appearances in the stool. This case also had an ankylostoma infection and the days on which eggs of this worm were present are shown in a separate column.

On the ninety-fifth and ninety-sixth days there was an attack of unknown origin. At this time blastocystis, which had been present

in small numbers before, were very numerous indeed. After the attack of diarrhœa had passed off, the blastocystis were again reduced. The table shows the presence or absence of *E. coli* free and encysted, eggs of ankylostoma, the character of the stool, and whether this was the result of a saline purge or not during a period of about four months.

Days	*E. c.c.*	*E. f.*	Ankylo-stoma	Saline	Stool	Days	*E. c.c.*	*E. f.*	Ankylo-stoma	Saline	Stool
1	−	+++	+	+	B.l.	43	−	++	+	+	B.uf.
2	+	++	−	−	B.uf.	44	−	−	−	−	B.f.
3	+++	++	+	−	B.uf.	45	++	−	−	−	B.f.
4	++	−	−	−	B.uf.	46	++	−	−	−	B.f.
5	++	−	−	−	B.f.	50	+++	−	−	−	B.f.
6	+	−	+	−	B.f.	52	−	−	−	−	B.f.
7	+	−	+	−	B.f.	56	+	−	−	−	B.f.
11	++	+	+	+	B.uf.	62	++	++	−	−	B.uf.
12	+	−	−	−	B.f.	63	−	−	−	−	B.uf.
16	+	−	−	−	B.f.	65	++	−	−	−	B.f.
17	+	++	−	+	B.uf.	68	−	++	+	+	B.uf.
18	+	−	−	−	B.f.	69	−	+	+	−	B.uf.
19	−	−	−	−	B.f.	74	−	+	+	+	B.uf.
20	−	−	−	−	B.f.	76	+	−	−	−	B.f.
21	−	−	−	−	B.f.	81	+	−	−	−	B.f.
22	−	−	−	−	B.f.	89	++	−	−	−	B.f.
27	+++	+	−	−	B.f.	90	++	−	−	−	B.f.
29	−	−	−	−	B.f.	91	++	+++	++	+	B.uf.
30	−	++	−	+	B.uf.	94	−	−	−	−	B.sf.
33	−	−	−	−	B.f.	95	++	++	+	−	B.l.
38	++	++	+	+	B.uf.	96	++	++	+	−	B.l.
39	−	−	−	−	B.f.	107	+	+	−	−	B.uf.
40	++	−	−	−	B.f.	120	−	−	+	+	B.uf.

B.l. = Brown liquid. B.uf. = Brown unformed. B.f. = Brown formed.
B.sf. = Brown semiformed.

(9) *Is E. coli ever Pathogenic?*

E. coli is the commonest protozoon parasite of the human intestine all the world over, and it is evident that for this reason it must be present in all kinds of intestinal disease. The statement made by Low that it is gradually being borne in upon him that *E. coli* is sometimes pathogenic in causing diarrhœa is of little value as it is backed by no evidence and he does not tell us how long he has been accustomed to control his cases, with a clear idea as to the differential diagnoses between *E. coli* and *E. histolytica*. Because one encounters *E. coli* in a diarrhœic condition one must

not be misled into the notion that it is the cause of the trouble. In Egypt the largest infections of *E. coli* we have encountered, and some of these very large indeed, have been in perfectly healthy men with normal stools. Further, taking into consideration a large series of hospital cases with diarrhœa the percentage of *E. coli* infections is considerably below that of healthy men in the same locality. The cases which have intestinal symptoms of diarrhœa with an *E. coli* infection invariably retain the *E. coli* infections when they recover, and as will be seen from the records of the *E. histolytica* infections treated by us, the *E. coli* infection almost invariably remains after the *E. histolytica* has disappeared. Of greatest importance, however, from the point of view of the pathogenicity of *E. coli* are the cases of infection which have been followed for very long periods. It is impossible to state that these individuals never have attacks of diarrhœa at any time, for who does not at some time or another during the course of every year, especially in a warm climate? But it can be definitely asserted that they show no abnormal tendency to diarrhœa. There is, therefore, in our opinion no justification for regarding *E. coli* as pathogenic.

A note of warning may perhaps be sounded for the benefit of those medical men to whom the subject of the intestinal protozoa of man is a new one. Those who have not been accustomed to study these organisms in any detail, and have only recently begun clearly to differentiate between them, must not be led astray by their comparative attractiveness and large size. The great majority who examine for intestinal organisms do so only in the case of sick people who have some intestinal disorder, and they have no means of comparing their findings with what occurs in healthy individuals. Accordingly it seems quite natural to them to attribute to the intestinal protozoa, both amœbæ and flagellates, which are comparatively easily recognized, pathogenic powers which they may not possess. It seems to us that there is only one possible plan of arriving at a safe conclusion as regards the relative frequency of protozoa in healthy and sick people, and it is one which is hardly practicable on a large scale. It is to take a large series of healthy individuals and to examine their stools for about one week while they are being purged by means of salines and to compare the results thus obtained with those from a similar series which are suffering from diarrhœic conditions acquired naturally. In this manner the relative incidence of some of the amœbic and flagellate infections in healthy and diarrhœic subjects might be obtained.

(10) *Lamblia intestinalis.—Reproducing Forms.*

(Plate II, figs. 1 to 5.) *

It was suggested by one of us (C. M. W.), who had made observations on these flagellates both in man and animals during a period of many years, that *L. intestinalis* possibly multiplied only in the encysted stage. This suggestion was put forward because free dividing forms had been constantly looked for, but never found even in animals which were killed and examined for the purpose. It was perfectly understood that this view was at variance with what we know of other intestinal protozoa, but, as *L. intestinalis* is a parasite of the small intestine, it was thought that forms which encysted high up in the gut might still be liberated under the action of the pancreatic juice a little lower down when division within the cyst had been accomplished. In his criticism of this view (*British Medical Journal*, June, 1916) Woodcock seems to forget that lamblia lives in the small intestine, and that in order to reach the pancreatic fluid there is no necessity for the cysts to escape from the intestine and be swallowed by the same or another host, as must be the case with protozoa which live only in the large intestine.

The rarity of dividing forms in the stool may be due to the distance of the lamblia from the rectum, but Woodcock offers no explanation of the fact that direct examination of the small intestine of infected animals fails to reveal dividing forms, nor does he tell us if he has searched for them in this situation. One would naturally conclude that dividing forms existed, and it was only with reluctance after many fruitless searchings that another view was put forward. It still has to be proved that the view is incorrect, though observations we have recently made on the case to be described now show that, at any rate under certain conditions, lamblia may divide as a free-living flagellate.

A patient was admitted to hospital with diarrhœa and the passage of large quantities of mucus. Examination of the stool revealed lamblia in enormous numbers, the mucus portion of the stool being simply packed with free-living flagellates. The patient was also a carrier of *E. histolytica*. Films were fixed in Schaudinn's fluid and stained with iron hæmatoxylin. The lamblia were beautifully preserved, and examination of the stained films showed all stages of undoubted binary fission. The dividing process, as illustrated in the figures (Plate II, figs. 1 to 5),* could

* *See inset between pages* 148 *and* 149.

be traced from the commencing division of the nuclei to the completion of longitudinal splitting of the flagellate.

In this process the whole complicated system of sucking disc with its related flagella is reproduced on the dorsum of the flagellate, but the tracing of the details of this process is a very difficult matter which is only to be compared with the difficulty of following the division in the encysted forms. When the whole flagellar apparatus has been duplicated the subsequent division can be readily followed, and is so clearly illustrated in the figures that little description is necessary. Apparently the flagellate splits longitudinally, the fissure passing in between the sucking discs from before backwards. Before this takes place the complete apparatus of the new flagellate is reproduced. The final stage is seen where two flagellates are attached only by the tapering posterior extremities.

Division of the flagellates had evidently been taking place with rapidity, for there was a very great variation in the size of those present in the film. There were present flagellates showing every gradation in size, from large forms measuring 17 by 10 microns to smaller forms measuring 10 by 6 microns. In some parts of the film streaks or patches of mucus occurred which were packed with these small forms, while in other places the mucus contained only large forms. The small flagellates had the same structure as the larger ones, except that the sucking disc appeared to occupy a greater portion of visible surface of the flagellate. There was no tendency for these small forms to take on the octomitus structure, so that there is no reason to assume, as some have done, that the lamblia is a later stage in the development of octomitus. An octomitus of man has recently been described by Chalmers and Petskola in the Sudan. Kofoid and Christiansen have recently described binary and multiple fission in the lamblia of mice (*Proc. Nat. Acad. Sci.*, November, 1915).

As regards the cysts of lamblia we have little new information to offer. In certain cases where the cysts had apparently been formed only recently, judging by the thinness of the wall and the fact that they had not yet assumed the perfectly ovoid form so characteristic of the mature cyst, the single lamblia within could still be seen moving its posterior extremity slightly from side to side, while the central pair of flagèlla were undulating slowly as one often sees them in lamblia which have ceased to swim about actively. At a later stage the cyst becomes more accurately ovoid and the wall tougher, while the single lamblia within may or may

not have divided. We have not obtained any evidence in support of the view that the cysts are formed to enclose two conjugating individuals. Whether such cysts occur or not is still undecided, but it is perfectly clear that the majority of the cysts are formed round single individuals.

(11) *Course of Lamblia Infections.*

It has already been shown that lamblia infections may persist for several years, but during the course of such an infection the flagellates or their cysts are not always to be found in the stools. The cysts alone are usually found, and judging by these the infection fluctuates, there being periods when the cysts are very numerous and others when they are absent. We do not know whether this absence of cysts from the stool means that the flagellates in the small intestine are reduced in number, or whether there has been a sudden cessation of cyst production. Nor do we know the cause of this change. In watching the lamblia infections from day to day a case, which has been passing a + + + infection of cysts regularly for a fortnight or longer, will suddenly show a reduction to + +, then to +, and then entire absence of cysts. Just as suddenly after an interval of a week or more a few cysts will be found and the infection will work up again to a large one. The course of several lamblia infections is shown in the charts illustrating the cases of *E. histolytica* and flagellate infections accompanying this paper.

Pathogenicity of Lamblia.—Of all the flagellates of the human intestine lamblia has the best claim to pathogenicity. This does not mean that it always produces symptoms, nor that every one with such an infection is to be isolated and treated as a dangerous carrier. Such a course would be obviously absurd in time of war, for there occur many healthy, able-bodied men who are carriers of lamblia for every one who shows symptoms or is incapacitated in any way by its presence.

The reasons for regarding lamblia as pathogenic are not merely the presence of the flagellates in diarrhœic conditions, but the fact that certain cases of lamblia infection have attacks of a characteristic nature when they pass large quantities of yellowish mucus in which occur unencysted lamblia in myriads. A small portion of such mucus may show the flagellates lying over one another and filling the entire microscopic field. Such attacks of diarrhœa with mucus occur at intervals, and whenever the attack comes the same mucus is passed with the same microscopical appearances. A case

of this kind was described by one of us (C. M. W.). A man had repeated attacks of this kind during several years. It is difficult to explain the presence of the large quantity of mucus on the occasions of the attacks, and above all the crowding of the mucus by the flagellates, without assuming that the mucus must be produced by the intestine at the site of the lamblia infection and that the flagellates are directly responsible for its production. We thus have much more proof of the pathogenicity of lamblia than in the case of either trichomonas, tetramitus, or any of the rarer flagellates, all of which are most usually encountered in diarrhœic conditions in which mucus production is not generally a feature of the case.

(12) *Tetramitus mesnili.—Free and Encysted Forms.*

(Plate II, figs. 6 to 18.) *

This flagellate is of very common occurrence and has been more frequently found since we have been able definitely to recognize the encysted form which is at times the only stage met with in the stool.

Now that it is being generally recognized, the flagellate will undoubtedly prove to be of world-wide distribution. It has certainly often been mistaken for trichomonas and possibly other organisms, though attention to details of structure should prevent any such errors in the future. One of us (C. M. W.) has noted tetramitus in iron hæmatoxylin-stained films of fæces from the Philippines and Panama, two places from which the flagellate has not hitherto been recorded. In these places the flagellate must have been seen and described under other names. The flagellate is typically pear-shaped, though very frequently it is somewhat flattened or even leaf-like, while the long-drawn-out flattened posterior extremity may be twisted and folded in a variety of ways to produce peculiar grooves and spiral turns. The figures represent some of these twisted forms which are so common that they can hardly be called abnormal. (Plate II, figs. 6 to 9.) *

It seems most probable that *Tetramitus mesnili* multiplies as other intestinal flagellates by binary fission, but we have not yet met with the dividing stages.

We have, however, been able definitely to recognize the encysted forms of this flagellate, and these are of importance because an infection can be recognized by them just as a lamblia infection can

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* *See inset between pages* 148 *and* 149.

be identified by the presence of its cyst in the formed stools from which the flagellates are usually absent.

The cysts of *T. mesnili* were first described by one of us (C. M. W.) in the original description of the organism. The cyst was there depicted as a small oval structure very much like a small lamblia cyst, within which could be detected the nucleus and cystostome of the flagellate. Though this type of cyst occurs not infrequently, the commonest cyst is distinctly pear or bottle shaped with one end narrower than the other as shown in the figures. This type of cyst was figured by Prowazek and Werner in a paper describing some tetramitus infections met with in Hamburg (*Arch. für Schiffs. und Tropen-Hyg.*, Bd. 18, 1914, Beiheft 5, Festschrift).

As seen in fresh preparations the pear or bottle shaped cysts are pale bodies which show no internal structure except two or three, rarely more, small refractile, greenish granules (Plate II, figs. 10 to 12).*

They vary in length from 7 to 10 microns and sometimes occur in such large numbers in the constipated stool that several can be seen in every field of the one-twelfth objective. The recognition of the cysts in the fresh condition is sometimes rendered difficult on account of the presence of a yeast of similar shape. The yeast, however, shows clearly its internal structure of granular cytoplasm with large vacuole, the whole being much greener than the pale structureless tetramitus cyst. The yeast varies in shape more than the tetramitus cyst and the characteristic budding forms are to be found if one looks for them (Plate II, figs. 16 to 18).*

The tetramitus cysts stain readily by the iron hæmatoxylin method after fixation in Schaudinn's fluid, and when suitably differentiated they show the characteristic nucleus and the cytostome rim (Plate II, figs. 13 to 15).* One or more granules are present near the narrow end of the cyst and the deeply staining line which borders the cytostome takes origin in one of these. In addition one can sometimes make out the flagellum, which normally lies within the cytostome also arising from this granule. Further than this little structure can be detected and we have not seen any indication of nuclear multiplication. The four-nuclear cyst figured by one of us (C. M. W., *Lancet*, November 27, 1915, fig. 17, and *Journal of the Royal Army Medical Corps*, December, 1915, fig. 17, p. 614), as a possible later stage in the development of the tetramitus cyst, has turned out to be the cyst of *E. nana* described on *page* 90 and not a cyst of the flagellate.

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* *See inset between pages* 148 *and* 149.

(13) *Course of Tetramitus Infections.*

On account of the characteristic cysts it is much easier to control a tetramitus infection than one of trichomonas. On those occasions when the flagellates are absent from a formed stool their presence in the gut is still detected by the pear-shaped cysts which escape in the fæces. Though, like trichomonas, tetramitus tends to be present intermittently in the stool, this feature is not nearly so marked. In case Howard, for instance, the flagellate was present practically continuously for over fifty days. In another case, Gildel, it was present for ninety-one days, being absent only for about a month at the middle of the observation owing to treatment by emetin orally administered. A glance through the cases of *E. histolytica* infections illustrated in the tables at the end of this paper will show how the tetramitus infection persists, and though it may disappear for a time it reappears again later. As such an infection can persist for over three months it must do so for longer periods, though we have not the same data as we have in the case of the lamblia infections, which one of us (C. M. W.) has already shown to be able to persist in the intestine for several years.

(14) *Trichomonas intestinalis and Trichomonas in the Mouth.* (Plate III, figs. 18 to 23.)*

The general structure of this flagellate has already been described by many observers, and it has been pointed out that the number of flagella is either three (trichomonas), four (tetratrichomonas), or five (pentatrichomonas). The commonest type seen by us has been the tetratrichomonas (Plate III, figs. 19 to 23),* though three and five-flagellar forms (Plate III, fig. 18)* have been seen, each on one occasion. Apart from the flagella the three types show no variations in structure, though a peculiar difficulty in the making of satisfactorily stained films of the human intestinal forms renders their study somewhat tedious. Very good preparations showing the main features can be made by exposure to osmic vapour of the wet film, which is then dried and stained by eosin azur. A good method is to expose a small drop of saline emulsion of fæces on a slide to the vapour of the osmic acid bottle for about ten to twenty seconds and then to be spread out thinly, dry and stain. It will be seen that all three types possess definite axostyles, the presence of which in the human intestinal trichomonas some observers seem to doubt. For instance, the figures given by Brumpt in his "Précis de Parasitologie" (p. 195) do not show

* *See inset between pages* 148 *and* 149.

axostyles. The structure, though most readily seen in the osmic vapour films, can, with careful examination in well differentiated films, be made out in iron hæmatoxylin preparations fixed in Schaudinn's fluid. As in other trichomonas, the axostyle appears to take origin in the neighbourhood of the basal flagellar granule. The structure of the undulating membrane with its attached flagellum and the basal supporting fibre call for no special remarks. In sublimate-fixed films the nucleus appears spherical with a fine nuclear membrane and centrally placed karyosome. The trichomonas in the human intestine are rarely above 10 microns in length so that their study is very difficult.

We have made most careful search, in a number of cases followed from day to day, for encysted forms, and though we have several possible structures in mind we have come to no definite conclusions on the matter.

It would seem that encysted forms must be present at some time or another, for we now know that all the common protozoa of the human intestine have such stages.

In one case (Morris) which was being controlled daily on account of *Waskia intestinalis* infection, *Trichomonas* sp. was found in the mouth along with entamœbæ in a pyorrhœa exudate. The case had been most carefully watched for many weeks and at no time were trichomonas found in the stool. The mouth trichomonas persisted, so that the patient must have constantly swallowed the flagellate, yet no intestinal infection was established. This observation would seem to suggest that the mouth trichomonas differs from the intestinal form as the mouth entamœba does. The mouth trichomonas in this case had three flagella and possessed a definite axostyle. Though the patient had a gut infection of both waskia and tetramitus, these flagellates though specially searched for were never found in the mouth.

(15) *The Course of Trichomonas Infections.*

In a former publication (*Lancet*, November 27, 1915) one of us (C. M. W.) wrote that trichomonas infections did not appear to be of very long duration, as the flagellates quickly disappeared from cases under observation. We have had in Egypt a better opportunity of studying the flagellate infections and it is quite evident that a trichomonas infection may persist for long periods, the parasites, however, only being present in the stool intermittently. They are present perhaps for a week and then disappear only to return later.

The difficulty of controlling such an infection is all the greater as encysted forms are not known. With lamblia and tetramitus for instance, infection can be recognized by identifying the encysted forms when the free forms are absent in the constipated or formed stools. With trichomonas, on the other hand, one very rarely finds evidence of infection other than in the soft unformed or liquid stools. Apart from the intermittency of the infection dependent on the character of the stool, there seems to be a real one when flagellates may be absent for days together even when the stools are quite soft or liquid. In one case (Howard) trichomonas was present practically continuously for over three weeks, when it vanished not to reappear during the following three weeks. This was the longest period we have observed of the continued presence of trichomonas in the stool. In another case (Pointer) trichomonas was only absent for a few days at a time during an observation of sixty-two days.

(16) *Pathogenicity of Trichomonas.*

As already explained, this flagellate is hardly ever found in any but soft or liquid stools, and it is this reason which has led observers to regard it as pathogenic. It is quite possible that sometimes a flagellate produces symptoms of diarrhœa resulting from an irritation produced by large numbers of organisms, but we are convinced that in the majority of cases of diarrhœa its presence is only accidental, and that it is fixed upon as the cause of trouble because it happens to be the most conspicuous organism in the stool. In an examination of 263 hospital cases where the stool was liquid trichomonas was only found in eight, while of 393 with soft unformed stools it was found in fourteen, and in 165 stools consisting of blood and mucus it was found in three. There is very little difference in any of these figures, though they are distinctly higher than the findings in healthy men given in an earlier part of this paper. This is only to be expected when one remembers that the percentage of liquid and soft stools among the healthy men is much lower than amongst the hospital cases. In the cases which had blood and mucus in the stool the trichomonas was certainly not the cause of the dysenteric symptoms, yet the liquid and soft stools did not show a definitely higher proportion of trichomonas cases. From these figures there is to be gathered nothing which will support the pathogenicity of trichomonas. In warm countries where practically every incoming white man suffers from periodic attacks of diarrhœa, either from indiscretions of diet, exposure, or other causes, it is not surprising that trichomonas

should occur in some of these. It is, however, obviously illogical to call all such cases of trichomonas diarrhœa, while there is no excuse whatever for the use of the term "trichomonas dysentery." The cases of dysentery in which trichomonas has been present have been cases of bacillary dysentery.

We are of the opinion, therefore, that while trichomonas may, in some cases, cause diarrhœa, and possibly a diarrhœa persisting over several months, in the vast majority of cases in which it has been found it has in no way been the cause of trouble. Those who are inclined to attribute to the flagellate a pathogenicity must never forget that there are hundreds of cases of diarrhœa both transient and chronic in which no protozoa whatever are to be found for every one in which trichomonas is present. The inclination to attach importance to it as a pathogenic agent is the direct outcome of its size and structure.

(17) *Coccidium* (*Isospora*).

As we have already mentioned, the isospora which one of us found fifteen times in the examination of 556 cases in London during the latter half of 1915, was found only once amongst the large number of men examined by us in Egypt during the first six months of 1916. It seems probable that the infection came from Gallipoli.

The single case (Webber) seen by us in Egypt was in an *E. histolytica* carrier who had been discovered as such in the routine examination of men in Sidi Bishr camp. The man was brought into hospital for treatment of his *E. histolytica* infection and the first oöcysts were seen on the eighth day that the stool was examined and on the day that a course of one-grain emetin injections was commenced. The twelve injections of emetin did not rid the man of the coccidium infection any more than of the *E. histolytica* infection. The oöcysts of the coccidium were few in number during the first few days of the course, but they became more numerous towards the end and were still more numerous after the course was finished. Nine days after the completion of the first emetin course a second course was given. This time the double treatment of injections and orally administered emetin ($1\frac{1}{2}$ grains a day) was employed. The oöcysts became fewer and were last seen on the sixth day of the course, the *E. histolytica* having vanished after the second day. The case was most carefully controlled for a month after treatment, several films being frequently examined but no recurrence of the oöcysts occurred. The treatment seemed not only

to have cured the patient of the *E. histolytica* infection but of the coccidium infection as well.

As regards the pathogenicity of the coccidium nothing can be gathered from this case, for though there was also an *E. histolytica* infection there were no symptoms attributable to either. The patient had been on the Peninsula, where he had had dysentery, and if the coccidium infection had been contracted there he must have carried it for seven months, as he had left in September, 1915.

As regards the oöcysts from this case, development was easily obtained, many of them completing their development in twenty-four hours. Some of the oöcysts showed a peculiar tendency to abnormal development in producing only a single sporocyst containing eight sporozoites. Developed oöcysts were given in large numbers to a young mouse but no infection occurred. Kittens fed on developed oöcysts also failed to become infected. The isospora of cats is very common in Alexandria, but the oöcysts are quite unlike those of the isospora of man.

(18) *Iodine Cysts* (*I-Cysts*).

(Plate III, figs. 12 to 17.) *

During the early months of the year these peculiar structures, with their strongly iodophilic bodies, were frequently met with in the stool. They were present sometimes in very large numbers and it is interesting to note that the most intense infections were met with amongst a series of native prisoners we examined in Hadra prison. They were present in 14·8 per cent of the prisoners.

Examination of the daily ration microscopically did not reveal any source of infection.

The I-cysts are generally easily recognized if one employs iodine. Sometimes without this it is impossible to identify them certainly, as the iodophilic body may be mistaken for the chromidial body of a cyst of *E. histolytica*. The iodophilic body, however, tends to be rounded or lobed, while chromidial bodies in the *E. histolytica* cysts are generally rod like. The I-cysts vary greatly in size and shape. They may be quite small with a diameter of 7 or 8 microns, or large with a diameter of 15 microns or more. Though most usually they tend to be circular or oval in outline they may be lobed or show processes which suggest an outgrowth into a filament. The single nucleus is smaller than the nucleus in

* *See inset between pages* 148 *and* 149.

the single-nuclear *E. histolytica* cyst, and, furthermore, it is different in appearance and structure. In one or two cases we have come across fairly large infections of cystic structures which resemble the I-cysts in every way except for the absence of the iodophilic body. These caused us a good deal of difficulty as there was a decided resemblance to *E. histolytica* cysts. After careful observation of the cases it was possible to make up one's mind that these were really I-cysts minus the iodophilic bodies. In typical I-cyst infections, where the majority of the cysts have the characteristic iodophilic bodies, a few may be seen in which these are absent (Plate III, fig. 15).*

In one or two cases there have occurred in the stool large spherical cysts which look very much like cysts of *E. coli* with a single nucleus. In stained films or iodine preparation each is seen to have a single nucleus, which is a spherical body with a very thick nuclear membrane, while the central part is clear and unoccupied by granules or other structures. It has the appearance of a homogeneous body with a central vacuole. When first seen it was thought the cysts were peculiar forms of the cysts of *E. coli*, but by following the cases for a few days it was found there was no tendency for recognizable stages of *E. coli* cysts to appear. It was evident that they were not amœbic cysts. It seems most probable that they represent large I-cysts which are devoid of iodophilic bodies or structures of an allied nature. One such case as this was seen by one of us during the routine examination in London of patients returning from Gallipoli at the end of 1915.

(19) *Blastocystis.*

These organisms are of exceedingly common occurrence, at least a quarter of the stools examined containing them. In certain cases they are present in very large numbers and the largest infections we have seen have been associated with an acute diarrhœa. Under these conditions one often notes abundance of very large forms quite as large and even larger than cysts of *E. coli*. On the other hand, there may be an overwhelming infection of very small forms. Whether large or small, it will be noted that under these conditions the blastocystis are mostly in various stages of division, suggesting that active multiplication is taking place.

Often the blastocystis occur in large clusters, and on one occasion such a cluster was enclosed in a definite cyst, reminding one of the multiple division stages described by Alexeieff (text fig. 5).

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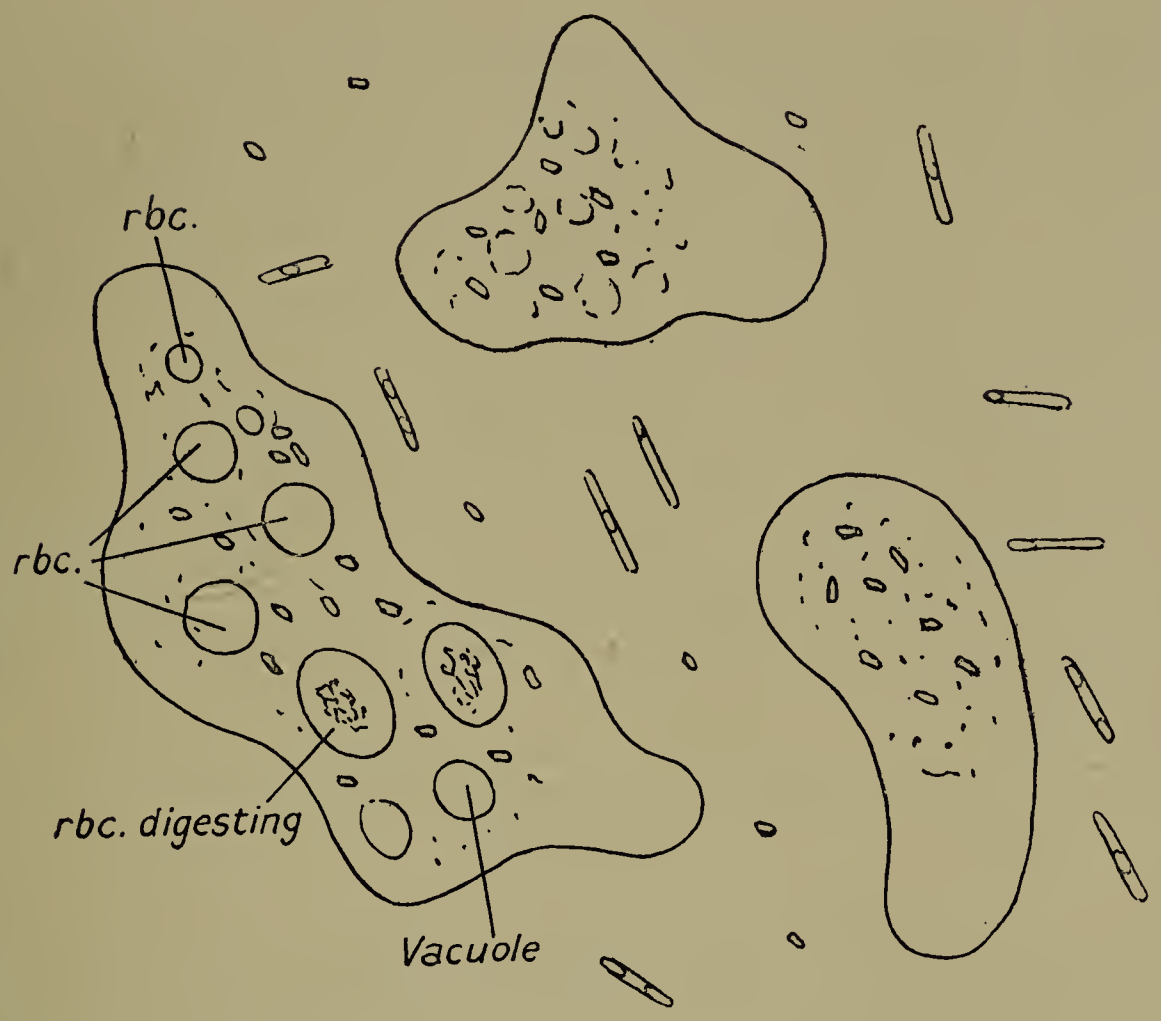
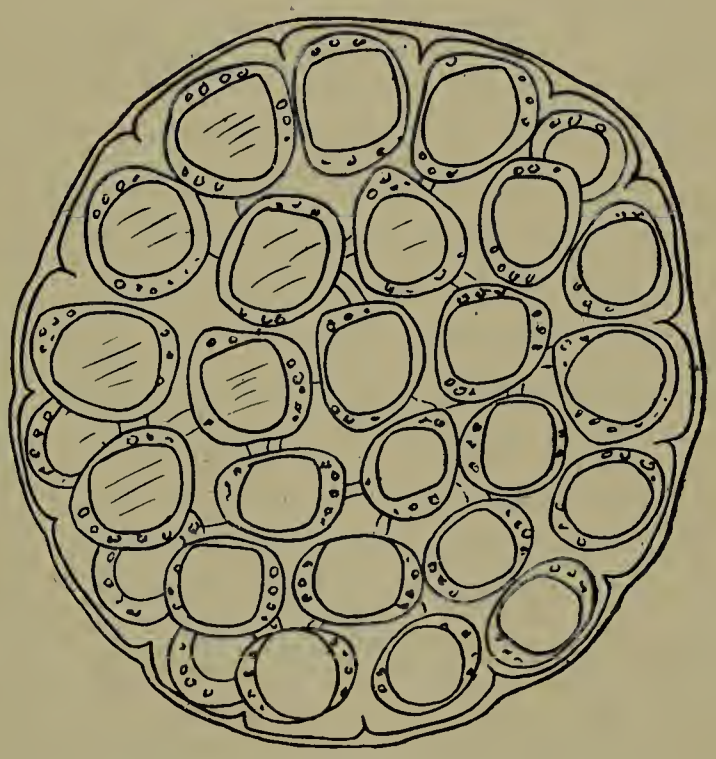
* *See inset between pages* 148 *and* 149.



TEXT FIG. 4.—A drawing of part of the field of the microscope showing *E. histolytica* containing spores of a spore-bearing bacillus (*B. megatherium?*) which was plentiful in the stool. One of the amœbæ contains several red blood corpuscles. Case Smith, July 14, 1916.

(*See page* 68.)



TEXT FIG. 5.—Multiple reproduction of blastocystis. Diameter of large cyst sixty microns. Case Wilkinson.

(*See page* 84.)

In one instance a large blastocystis infection developed in a case which was being controlled for *E. coli*. It was noted that though blastocystis were present it was only in small numbers. At one period these increased in numbers and there suddenly developed an acute diarrhœa, the stool then containing enormous numbers of dividing blastocystis. After the attack was over the blastocystis were reduced to their original small number.

The fact detailed above would suggest a possible pathogenicity for blastocystis when present in any quantity, but one has to be cautious in coming to such a conclusion, for all other factors must first be eliminated. For instance, in certain cases of undoubted bacillary dysentery the blastocystis have been very numerous in the stool while, large infections may be met with in perfectly healthy individuals.

(20) *Waskia intestinalis.*

(Plate IV.) *

The small flagellate for which we suggest the name *Waskia intestinalis* was found in two cases in Alexandria and there is some evidence that the second case became infected from the first while in hospital. The first case was one of a man who was admitted to hospital as a carrier of *E. histolytica*. The flagellate was first noted in the stool a week after his admission and was regularly present for one and half months. Towards the end of this period a tetramitus infection appeared, while the waskia infection became smaller and finally disappeared. The flagellate infection had withstood a course of injections of twelve grains of emetin early on in the observation. The second case was that of a man who was admitted to hospital also as a carrier of *E. histolytica*. He was passing the small type of *E. histolytica* cysts. On his admission to hospital the cysts became reduced in numbers and finally vanished from the stool, which was being carefully searched daily. After a period of three weeks in hospital the patient suddenly developed a large waskia infection, and it is interesting to note that he was in the same ward as the first case and occupied the next bed but one. It seems probable that the second case contracted the infection from the first case, who was passing enormous numbers of the encysted forms of waskia in the stool. The second case still infected was discharged from hospital ten days after the infection was detected. There was no evidence that the flagellate

* *See inset between pages* 148 *and* 149.

was in any way pathogenic. The first case tended towards constipation, which had to be relieved by repeated doses of salts. The second case was also treated in this way while in hospital.

Description of the Living Flagellate.—The living flagellate (Plate IV, figs. 1 to 6)* is a small active oval organism which dances about amongst the fæcal matter by means of its two flagella of different strength and action. The long, thin, anterior flagellum lashes about continuously and propels the flagellate through the liquid, while the stouter and shorter flagellum which projects from the cytostome may work either regularly, but at a different rate from the anterior one, or irregularly with periods of rest alternating with periods of activity. This independent action of the stout cytostome flagellum, especially when its action is intermittent, gives a peculiar jerky movement to the anterior end of the flagellate as it swims forward under the regular action of the long anterior flagellum. In cover-glass preparations the flagellate has a peculiar habit of applying itself to the surface of the cover-glass or slide to which it adheres. In this position the action of the two flagella can be easily studied, and it is in this side view attitude that the flagellate reminds one so forcibly of the body outline of a bird.

In shape the flagellate is ovoid with the anterior end rounded and the posterior end pointed. There is a cytostome at the side of the anterior end, while the tapering part of the posterior end tends to be on the side of the body opposite to that on which the cytostome opens. This arrangement makes the flagellate bilaterally symmetrical. From the anterior end, but slightly nearer the cytostome side of the body, there takes origin a thin flagellum which equals the body in length. A second stouter and shorter flagellum arises from the inner part of the anterior wall of the cytostome. It passes slightly backwards and outwards through the mouth of the cytostome, from which it projects for a considerable distance. The separate action of the two flagella has been explained above. The cytoplasm of the flagellate body is very pale, much paler and less refractile than that of small forms of tetramitus or trichomonas. It is frequently much vacuolated and the vacuoles contain bacilli or cocci which have been ingested. One can often see quite large bacilli entering the cytostome. The nucleus occupies the anterior end of the body and in the living flagellate can hardly be detected except as a clear, more homogeneous area which is devoid of the granulations or vacuoles of the rest of the body. The length of the body varies from 4 to 9 microns, the majority

* *See inset between pages* 148 *and* 149.

of flagellates being 5 to 6 microns long. The width varies considerably; the narrow forms 3 to 4 microns in width mostly resemble a bird in outline; wider forms occur in which this resemblance is lost, and finally forms which are practically spherical are found. In addition to these forms there occur others which are possessed of two cytostomes and two pairs of flagella. These might be either dividing or conjugating forms, but the examination of stained specimens shows the former view to be correct.

Encysted forms of the flagellate are common in the stool (Plate IV, figs. 7 and 8).* These are pear-shaped bodies 4·5 to 6 microns in length. They have a pearly-white appearance and are quite structureless. They remind one of small tetramitus cysts, but have not the few refractile granules which these cysts generally possess. That these bodies are actually cysts of the flagellates is proved by their constant association with it in the two cases mentioned and their complete absence from all other cases observed by us.

In the two cases of *Waskia intestinalis* infection the flagellates were present in the stool in enormous numbers during the height of their development; many flagellates were present in each field of the $\frac{1}{12}$ objective. In the first case, from the stool of which they disappeared after about six weeks' observation, the flagellates became reduced in number towards the end and appeared as if they were being crowded out by the larger and more vigorous tetramitus. Knowing, however, that flagellate infections are peculiarly irregular in their course, we recognize that it is possible that the infection had only been reduced temporarily.

Description of Stained Flagellates.—The flagellates were studied in films fixed in Schaudinn's fluid and stained by the iron hæmatoxylin method, and also in dry films stained by Romanowsky stain. In the stained film the flagellate is seen to have the same shape as that of the living organism. The nucleus at the anterior end can be more clearly seen, and is found to consist of a spherical nuclear membrane with usually a central karyosome. On the surface of the nuclear membrane towards the cytostome are two granules from which arise the two flagella. The dividing forms are seen to have a simple dividing nucleus in which a centrodesmose can be detected and at a later stage two distinct nuclei (Plate IV, figs. 9 to 14).*

The encysted forms show more details when stained than in the living condition. Certain nuclear structures can be made out

* *See inset between pages* 148 *and* 149.

within the cyst, though the exact significance of the appearances are doubtful. The nuclear membrane becomes much elongated and may stretch from one end of the cyst to the other, while the karyosome tends to become dumb-bell shaped and divided into several parts. Whether this is an indication of nuclear multiplication cannot be stated at present (Plate IV, figs. 15 to 20).*

The flagellate which has just been described from two cases has never been encountered before, and is evidently a new parasite of the human intestine, and for this reason we have given it the name *Waskia intestinalis*, after the Orwa-el-Waska section of the 19th General Hospital where it was first discovered.

(21) *Tricercomonas intestinalis*, n. g., n. sp.

(Plate III, figs. 1 to 11.)*

The small flagellate which we have found in about a dozen cases differs entirely from *Waskia intestinalis* already described. It is a small active organism which is very difficult to study on account of its rapid dancing movements. It has a spherical or ovoid body which is distinctly flattened on one side as in flagellates of the genus *Cercomonas*. The posterior end is drawn out and terminates in a flagellum which can be traced forwards along the flattened side to the anterior end of the body where three other long flagella originate. The tail flagellum appears to be attached to the surface of the body, and occupies this position in all the active movements of the organism. For this reason it seems clear that the flagellum is actually attached to the surface of the body and not merely applied to it as Woodcock thinks is the case in cercomonas, where the tail flagellum is similarly arranged. In the tricercomonas as well as cercomonas the cytoplasm of the body is drawn out into a tapering process which follows the flagellum for some distance, and it seems difficult to understand how this can be if the flagellum is merely applied to the body and not definitely attached. The posterior flagellum of tricercomonas and cercomonas is attached to the surface of the body as much as the flagellum of trichomonas is attached to the border of the undulating membrane, and in both flagellates this attachment may be broken down and the flagellum become free except at its point of origin. Tricercomonas, as the figures show, has three long anterior flagella and one posterior flagellum passing over the flattened side of the body, which moreover is sometimes grooved along its course. The flagellate resembles

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* *See inset between pages* 148 *and* 149.

cercomonas, except that it possesses three instead of one anterior flagellum. Hence the name tricercomonas which we suggest. In the fresh condition the flagellate is very active, but some time after leaving the intestines the movements are less violent and the details can be studied. No definite cytostome could be distinguished, though bacilli and cocci are ingested. The body changes its shape very readily, and is to a certain extent amœboid. The body of the flagellate measures 4 to 8 microns in length. The flagella are longer than the body. Sometimes the body of the flagellate is deformed, in that one or two curious pseudopodia or pedunculated processes project from the surface.

In one case the flagellate which was present in large numbers was associated with a small oval cyst 6 to 8 microns long by about half this in breadth. No structure could be seen in the unstained specimens, but in stained films the cysts were found to contain one, two, or four nuclei of a type resembling the nuclei of the stained flagellates. It seems very probable that they represent the encysted stage of the flagellate (Plate III, figs. 5 to 8).* In stained films the flagellate is found to have a nucleus like that of cercomonas, with a central karyosome and a nuclear membrane which is drawn out at one spot into a cone-like elevation, from the summit of which the flagella take origin. In stained films forms with two nuclei, probably dividing forms, can be found. As mentioned above the flagellate has been seen in about a dozen cases. Unlike *Waskia intestinalis*, which persisted in the stool for long periods, the *Tricercomonas intestinalis* was present only for a day or two at a time. In one case only was it present for as long as nine days, when it disappeared not to be found again. The cases were under observation in hospital and the stools were examined every day. There is no evidence whatever to suggest that the flagellate is in any way pathogenic. On account of its resemblance to cercomonas and its possession of three anterior flagella we suggest the name *Tricercomonas intestinalis*.

(22) *Entamœba nana*, n. sp.
(Plate I, figs. 10 to 23).*

The small amœba which, on account of its small size, we describe under the above name has turned out to be one of the commonest protozoa in the human intestine in Egypt, rivalling in some groups examined even *E. coli* in its frequency. The cysts

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* *See inset between pages* 148 *and* 149.

of *E. nana* were seen by one of us in cases in London during the latter part of 1915, but they were thought to be of a vegetable nature, or possibly a stage in the encystment of tetramitus. One of the four-nuclear cysts was figured in a paper on the "Human Intestinal Protozoa" (*Lancet*, November 27, 1915, and *Journal of the Royal Army Medical Corps*, December, 1915, fig. 17) as a possible later stage of development of the cyst of tetramitus, which was also present. The small free amœbæ were also met with on several occasions but were regarded as *Amœba limax*, though a failure to obtain a culture on agar media on which *A. limax* was growing threw some doubt on this. In Egypt, however, we have had ample opportunity of studying this amœba in cases which have been under daily observation in hospital and we have been able to definitely associate the small oval or spherical cysts with the small amœba. The type of infection with *E. nana* resembles that of other intestinal protozoa. The amœbæ or their cysts may be passed continuously over comparatively long periods, or the infection may be apparent for only a few days and then vanish only to reappear again after an interval. *E. nana* occurred in a good number of the cases which were being controlled in hospital for *E. histolytica* infections (*see* Tables of Cases, *pages* 160–177, etc.).

Very striking are the cases where cysts only in large numbers are passed daily in a constipated stool. The administration of a saline purge to such cases produces enormous numbers of minute amœbæ. In the soft unformed stool it is usual to find the free amœbæ and the cysts associated.

One must be careful to distinguish the cysts of *E. nana* from the cysts of the smaller strains of *E. histolytica*. The resemblance may be very striking and at times it may be necessary to stain films in order to arrive at a diagnosis. The small *E. histolytica* cysts are generally spherical or nearly so, while the cysts of *E. nana* are typically oval, very much like small lamblia cysts, though internally no structure can be detected. Spherical and irregularly shaped cysts of *E. nana* also occur, but these are always associated with the more typical oval forms. The cysts are never bottle-shaped like the cysts of tetramitus, with which confusion might take place.

Free Amœbæ.—The free-living amœbæ are very small and have a diameter of 5 to 10 microns. Their structure varies considerably. They may be very vacuolated and contain bacteria and cocci or they may appear quite homogeneous (Plate I, figs. 10 and 11).* The

* *See inset between pages* 148 *and* 149.

latter are possibly encysting forms which will give rise to the cysts which appear almost structureless when seen in the fresh condition. As a rule the amœbæ move sluggishly, throwing out one or more blunt ectoplasmic pseudopodia. They do not glide along in the true "limax" manner. The nucleus with its large chromatin block can sometimes, though rarely, be detected in the living amœbæ. The stained amœbæ (Plate I, figs. 12 to 17)* show the same clear or vacuolated cytoplasm seen in life. The structure of the nucleus can however be more clearly seen. Generally this appears to be of the "karyosome" type with a clear nuclear membrane and large centrally arranged chromatin block or karyosome which is often irregularly shaped. The position of the chromatin block at the centre of the nucleus is often only apparent, for in certain positions of the nucleus it is seen to be lying laterally against the nuclear membrane. In those cases in which it seems to be centrally situated the appearance may be due to the nucleus lying with the chromatin mass on the upper or lower surface of the nuclear membrane as the amœbæ lies on the slide. Sometimes the laterally placed chromatin mass is connected with a filament which passes across the nucleus to a granule lying on the nuclear membrane opposite to it (Plate I, fig. 15).* The occurrence of this type of nucleus in the amœbæ is of importance, for it is a very common type of nucleus of the encysted forms and greatly strengthens the belief that the cysts and the amœbæ are one.

Encysted Amœbæ.—The cysts of *E. nana* are oval, spherical or more irregularly shaped structures having a diameter of 7 to 8 microns when spherical and a length of 8 to 10 microns when elongated (Plate I, figs. 18 to 23).* The later type is most easily recognized as a pale whitish and structureless body in which no detail can be detected. Even when mounted in iodine it is only occasionally that the internal nuclei can be seen. The cysts of the amœbæ have hitherto been regarded as of a vegetable nature. Fixed in Schaudinn's fluid and stained with iron hæmatoxylin the nuclear details can be as clearly seen as the small size of the cysts permit. There are either one, two or four nuclei, and these are generally grouped together towards one end of the cyst. Most important is the structure of the nucleus. It will be seen by reference to the figures in Plate I* that the chromatin is generally arranged as a mass at one side of the nuclear membrane, while very frequently a fine filament connects the mass with a granule on the opposite

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* *See inset between pages* 148 *and* 149.

side of the membrane. The occurrence of this type of nucleus both in the free amœbæ and the cysts dispels all doubt as to the identity of the two.

As already stated, the *E. nana* is of very frequent occurrence, and has been constantly encountered in the course of routine examination now that we have learned to identify it. The amœba has undoubtedly been seen before, but has been confused with small forms of *E. coli* or *E. histolytica*, with which it is often found. It has only been by careful study of a number of cases, and above all of cases from which the other entamœbæ were absent, that we were able to exclude any connection with them. The course of the infection was very strikingly followed in two cases which had been cured of *E. histolytica* infections. The greatest amount of confusion has probably existed between this amœba and *Amœba limax*, or the amœba which is generally known by this name. *A. limax* can be cultivated from the human stool, but all our attempts to obtain culture of *E. nana* have failed. One stool which was much delayed in reaching us contained numerous minute amœbæ and small spherical cysts like the cysts of the amœbæ which readily grow on agar and which are quite unlike the cysts of *E. nana*. This material planted on agar plates gave rise to a good culture of amœbæ, and the spherical cysts were there reproduced. Material containing *E. nana* was inoculated on the same medium on the same day from two cases, but no growth was obtained. It seems clear, therefore, that *E. nana*, like *E. coli* and *E. histolytica*, will not grow on agar, which is suitable for the growth of *A. limax* or similar forms. The amœba which we have designated *E. nana* corresponds with none of the amœboid organisms which have been described from the human intestine. The *E. butschlii* of Prowazek, a much larger form, resembles it most. This author's description, however, is too meagre to allow of any comparison being made. Moreover, he does not describe any encysted forms.

As regards the nomenclature of this amœba we have placed it with the entamœbæ because it appears to be truly parasitic. In nuclear structure, however, there are certain differences, though, as with the entamœbæ, the chromatin material is mostly arranged on the nuclear membrane. Aggregations of chromatin are not uncommon in the nuclei of *E. histolytica*. Furthermore, it is very doubtful if nuclear structure alone can be regarded as a distinguishing feature of the genus Entamœba. It seems safer, therefore, to include this form with the truly parasitic amœba under the name *Entamœba nana* than to class it with the free-living non-parasitic amœba.

Summary of Matter discussed in Part II.

(1) It is often quite impossible to distinguish unencysted forms of *E. histolytica* from *E. coli*. The cysts, however, can be distinguished. Accordingly, it is safer to diagnose *E. histolytica* only if the amœbæ contain red blood corpuscles or if definite four-nuclear cysts are present. Some rules to act as a guide to the diagnosis of intestinal amœbæ are given.

(2) The cysts of *E. histolytica* vary greatly in size. There occur certain strains of *E. histolytica* with cysts 7 to 10 microns in diameter, strains with cysts of intermediate size and finally large strains with cysts 15 to 19 microns in diameter.

(3) There is no doubt that the presence of the four-nuclear cyst is an indication of infection with *E. histolytica*, even when there has been no history of previous dysentery. The cysts from carriers who have never suffered from dysentery may give rise to acute amœbic dysentery in kittens.

(4) Cases may remain infected with *E. histolytica* for years. There are in these cases periods of acute dysentery, when amœbæ with included red cells are present in the stool, alternating with periods of comparative health when only small amœbæ and cysts are passed. Cases may remain as healthy carriers for long periods without showing any signs of dysentery. It is probable that these healthy carriers really have some ulceration of the large intestine, which, however, is not so extensive as that in the acute dysenteric.

E. histolytica may establish itself in the gut with the production of true dysentery or without the production of any symptoms whatever. In the removal of new troops from England to areas in which amœbic dysentery is not endemic it is inadvisable to station them, even for a short time, in centres of amœbic dysentery like Egypt unless urgent military requirements leave no other alternative.

(5) The bacillary dysentery stool when seen at the height of infection is characteristic, both macroscopically and microscopically, and can be recognized by its general appearance and by the extraordinary cellular exudate when viewed under the microscope. The amœbic dysentery stool contains darker blood and mucus but cannot be recognized with certainty apart from the amœbæ. There is absence of the cellular exudate of the bacillary dysentery mucus.

(6) The characters of the unencysted *E. coli* are so indefinite as to render its identification, apart from the cysts, most difficult. We have obtained no evidence that *E. coli* can ingest red cells.

The cysts of *E .coli* vary in size from 13 to well over 30 microns. These cysts may contain chromidial bodies.

E. coli is not always to be found in the stools of persons who are known to be infected. There is no evidence that *E. coli* is even pathogenic.

(7) *Lamblia intestinalis* reproduces by a process of longitudinal division in the unencysted state. The flagellate may vary very much in size. Judging by the presence or absence of encysted or free forms of lamblia in the stool, the infection runs a very irregular course. The flagellates may be absent from the stool for comparatively long periods. Of all the flagellates of the human intestine lamblia has the greatest claim to pathogenicity.

(8) *Tetramitus mesnili* occurs in the stool in the free and encysted stages and one or both of these may be present at one time. Tetramitus infections, like those of lamblia, run a very irregular course.

(9) The commonest form of human trichomonas in Egypt is that with four anterior flagella (tetratrichomonas). The three (trichomonas) and five (pentatrichomonas) flagellar forms also occur. An axostyle is always present. A trichomonas of the mouth in pyorrhœa occurs and appears to be distinct from the intestinal form. Trichomonas infections may persist for long periods, during which the flagellates cannot always be detected in the stool. There is little evidence of the pathogenicity of trichomonas.

(10) A case of coccidium (isospora) infection was followed for some time. Emetin appeared to cure the infection.

(11) Iodine cysts (I-cysts) were commonly found and their likeness to cysts of *E. histolytica* was sometimes very close.

(12) Blastocystis infections were very common and the largest infections and the largest forms were found in cases of diarrhœa or dysentery.

(13) Two new human intestinal flagellates (*Waskia intestinalis* and *Tricercomonas intestinalis*) and a new amœba (*Entamœba nana*) are described.

PART III.*

Treatment of E. histolytica and other Protozoal Infections of the Human Intestine.

This part of the report is devoted to a consideration of the action of drugs on the various intestinal protozoal infections met with in man in Alexandria. The chief objective was the treatment and cure of *E. histolytica* infections, but as many of them were mixed with other protozoal infections it was possible to watch on these also the action of emetin or other drugs employed. The various protozoa, as regards their behaviour under treatment, will be now considered, while the charts showing the courses of the various infections in the individual cases treated appear at the end of the report.

(1) *Treatment of Entamœba histolytica Infections.*

The line of treatment adopted for *E. histolytica* infections has been almost exclusively the administration of emetin hydrochloride either by subcutaneous injection or by the mouth. In a few cases we have given methyl emetin sulphate which was prepared by Dr. Pyman, Director of the Chemical Research Laboratories, Wellcome Bureau of Scientific Research, and kindly placed at our disposal by him. One case which resisted emetin treatment was given treatment by pulv. ipecac. and later by thymol.

The majority of the cases have been healthy carriers who were mostly discovered during the routine examination of men in camps as explained earlier in this paper. Some of these were encountered during the examination of hospital cases which had been admitted for various reasons. In addition to the carriers a smaller number of cases of actual amœbic dysentery were treated, and by amœbic dysentery we mean a condition associated with the occurrence of blood and mucus in the stool with the presence of amœbæ showing definite included red blood corpuscles. We have included no doubtful cases in this list, such as might possibly be bacillary dysentery with free forms of *E. coli*, but all have been undoubted cases of *E. histolytica* infection which have been diagnosed according to the system explained in another part of the paper.

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* Reprinted from the *Journal of the Royal Army Medical Corps*, April–May, 1917.

The cases have all been treated as hospital patients with the exception of four, who continued their duties as usual during the course of emetin.

The stools were in almost every instance examined for a few days before treatment was commenced in order to obtain some idea of the course of the infection before an attempt was made to get rid of it, and a careful record was kept of the kind of stool passed and the various other protozoal infections present besides the *E. histolytica*. While the patients were in hospital at least one entire stool was inspected each day, an arrangement having been made whereby the stools passed into bed-pans were brought at once to a lavatory near the laboratory which was set apart for this purpose. After the course of treatment was completed the patients were sent to a convalescent camp and were quartered in a special section. Here they were given ordinary diet and were placed on light duty. Stools were collected from them on alternate days. As will be seen from the records of the cases at the end of the paper it was possible in this way to follow exactly the effect that treatment had upon the infection and to note when any return of this occurred.

Each case was kept under control for at least one month after the completion of the course of emetin before being discharged as cured. Of course an unavoidable fallacy entered into the control, and that was the impossibility of excluding the chance of reinfection. That infection might occur in the convalescent camp is shown very clearly in some cases by the fact that certain protozoa not hitherto present in the stools appeared a few days after the patients had joined the camp. That this may have occurred equally well with *E. histolytica* we recognize quite clearly, but if it has done so in any case it can only have had the effect of making our results appear worse than they actually were.

In carrying out the treatment we have had direct control of all the cases owing to the kindness of the hospital authorities in placing beds at our disposal for this purpose. The temperature and pulse-rate of the cases were taken regularly and a careful watch was kept for any signs of heart irregularity which might be attributable to the emetin which was being administered. A record was kept of the number of times patients vomited after emetin was administered by the mouth.

The observations on the series of cases here recorded have occupied about six months and have entailed an enormous number of stool examinations, as can very readily be seen from the protocols

of the cases, but the results we have obtained throw light not only on the course of the *E. histolytica* infections and the effect emetin has on these but has brought out many other interesting points in connection with other protozoal infections of the human intestine.

(*a*) *Treatment by Emetin Hydrochloride.*—The emetin used in these observations was the tabloid product of Messrs. Burroughs Wellcome & Co. For injection purposes it was dissolved in the strength of one grain in one cubic centimetre of distilled water. For oral administration we employed the same product, one grain dissolved in tinct. opii fifteen minims, or the half-grain keratin-coated tabloids. Emetin administered subcutaneously was always given in one single injection daily, as this causes far less worry to the patient, who naturally does not like to have his injections unnecessarily multiplied, and, furthermore, it saves the labour of those who have to give the drug. It has yet to be proved that two injections of half a grain a day are better in any way than a single injection of one grain. The cases we have treated fall naturally into three groups according to the method of emetin administration. In Group I the cases were in hospital with four exceptions. They were not kept in bed entirely but were allowed about during the greater part of the day. They were given hospital chicken diet and were treated by the administration of a single injection of one grain of emetin a day for twelve days, while the bowels were kept loose by a mixture containing sodium sulphate one dram taken three or four times a day.

In Group II the cases were treated in the same manner as those in Group I, with the difference that the drug was given by the mouth instead of by injection. The emetin was dissolved in tinct. opii one grain in fifteen minims, and this was given each night in a cup of tea. The salines were not given so regularly, as the emetin by the mouth tends to keep the bowels loose.

In Group III the cases were confined strictly to bed, were kept on milk diet and were given $1\frac{1}{2}$ grains of emetin a day for twelve days (one grain injection each morning and half a grain in keratin-coated tabloid by the mouth at night). In this group of cases salines were not administered regularly but were given if there was any sign of the stool being formed. In addition to the cases which fall into these three groups there are others which were treated differently, and these will be described in due course.

In the early part of our investigation the cases were treated as in Groups I and II, and, as the tables show, certain cases relapsed,

and some of these were re-admitted to hospital and treated as in Group III. In recording the results of the different methods of treatment it follows that such cases will appear in two tables, for it has happened that where treatment as in Group I has failed to bring about a permanent or even temporary cure, a subsequent treatment by another method has caused the infection to disappear in many cases.

Group I.—Cases treated by Emetin Injections of One Grain a Day for Twelve Days. Table X.

As already explained, these cases were given an injection of one grain of emetin a day for twelve days. The reason for adopting this line of treatment and the arbitrary limit of twelve days was that one of us (C. M. W.) made a detailed study of a case which was under his care and was treated in this way with a successful result in London last year at the Wellcome Bureau of Scientific Research. The case was one of a carrier of *E. histolytica* who was passing cysts after having had an attack of dysentery in the Sudan. It was realized that the case afforded a good opportunity of watching the course of such an infection and studying the effect of emetin on the carrier. The case was carefully controlled by repeated examination of the fæces and, finally, it was decided to give the patient a course of emetin. The actual injections were kindly given by Dr. G. C. Low in the Laboratory at the Wellcome Bureau of Scientific Research, while the control of the infection was carried on carefully as before. It was noted that the infection disappeared after the second injection of emetin and that there was no recurrence after a long period of control. The findings of the one of us (C. M. W.), who had gone abroad on active service, and who had made the very careful observations on this case, were subsequently published by Dr. G. C. Low (*Journ. Trop. Med. and Hygiene*, February 1, 1916).

As the case just mentioned had responded so successfully to this line of treatment it was decided to try it on a more extended scale, and this was the first method adopted by us in the series of cases recorded here. The cases treated and the results obtained are arranged in Table X in two main groups: (A), carrier cases which were passing cysts of *E. histolytica* and amœbæ without included red blood corpuscles, in stools free from blood and mucus, and (B), acute cases which had actual dysentery and were passing amœbæ with included red blood corpuscles.

TABLE X.—Emetin Injections One Grain a Day for Twelve Days.

(A)—Carrier Cases not showing any Amœbæ, with included Red Blood Corpuscles.

Name	A	B	C	D	F	G	Other infections: Before end of emetin course	Other infections: After end of emetin course
Mounsey[1]	++	CR	8	30	+	+	Tet.	E.c., Tet.
Smith, H.[2]	++	CR	4	30	+	+	E.c.	E.c.
Ure	+++	CR	3	35	+	+	—	—
Morgan, S.	+	CR	5	30	+	—	E.c., L., Trich.	E.c., Trich.
Hancock	++	CR	4	35	+	+	—	—
Thompson	+++	CR	4	34	+	—	E.c., L., Tet.	E.c., L., Tet.
Jones	+++	CR	1	32	+	—	E.c.	E.c.
Wynne	+++	CR	6	34	+	—	Tet.	—
Osgood	+	CR	1	25	+	—	E.c., Tet., E.n.	Tet., E.n.
Turnbull	++	CR	3	32	+	+	E.c., Tet.	E.c., Tet.
Burroughs	++	CR	4	30	+	—	E.c.	E.c.
Morris	++	CR	11	39	+	—	E.c., E.n., W.	E.c., E.n., Tet., W.
Wood[3]	+++	CR	2	27	+	—	E.n., Trich.	E.c., Trich.
Harris[11]	+	CR	13	29	+	+	E.c.	E.c., Tet., Trich.
Russell, F.	+++	CR	2	31	+	—	—	E.n.
Walker	+++	CR	2	10	—	—	E.c.	E.c.
Myers	++	CR	2	30	—	—	E.c., Tet., Trich.	E.c., Tet., Trich., T.c.
Pointer	++	CR	7	40	—	—	E.c., E.n., Trich.	E.c., E.n., Trich.
Harding	+	CR	?	32	—	—	E.c.	E.c.
Webb	+	CR	?	36	—	—	E.c.	E.c.
Cox	++	CR	3	32	—	—	Tet.	E.c., E.n., Tet.
Bowers	++	CR	7	30	—	—	E.c.	E.c.
Noon	++	CR	2	31	—	—	—	E.c.
Turner	+	CR	8	27	—	—	L.	—
Nicholson	+++	CR	8	30	—	—	—	E.c.
Page	++	CR	7	31	—	—	L.	E.c.
Beardwood	++	CR	2	28	—	—	E.c.	E.c.
Inkpan	+	CR	3	38	—	—	Trich.	—
Southgate	+	CR	1	35	—	—	—	E.c.
Nixon	+	CR	5	36	—	—	E.c., E.n.	E.c.
Neale	+	CR	2	31	—	—	Trich.	Trich.
Flynn	+++	CR	9	31	—	—	E.c., E.n.	E.c., E.n.
Kitson	+++	CR	2	32	—	—	E.c.	E.c., E.n.
Knight	++	CR	1	30	—	—	—	—
Ormrod	+++	CR	2	32	—	—	E.c.	E.c.
Cooper	+++	CR	1	35	—	—	E.c.	E.c., E.n.
Badham[4]	++	CR	3	50	—	—	E.c.	E.c.
Webber[5]	+++	R	9	1	+	+	E.c., Coc.	E.c., E.n., Coc.
Pero[5]	+	R	5	7	—	—	E.c., Tet., Trich.	Tet. Trich.
McQuade[5]	++	R	2	22	—	—	Tet.	Tet.
Squires[5]	+++	R	1	10	—	—	E.c., E.n., L.	E.c., E.n., Tet.
Boyd[5]	+++	R	10	8	—	—	Tet.	E.c., L., Tet., T.c.
Allen[6]	++	R	2	12	—	—	E.c.	E.c.
Cox, A.[6]	+++	R	6	24	—	—	E.c.	E.c., E.n., Trich.
Jackson, A.[6 11] ..	+	R	3	15	—	—	Tet.	Tet.
Main[6 11]	++	R	2	8	—	—	Trich.	Trich., E.c.
Ealdon[6]	++	R	5	11	—	—	—	E.c.
Spiers[7]	+++	NR	—	—	+	+	—	—
Healy[12]	+++	NR	—	—	+	+	—	—
Kettlewell[7]	+++	NR	—	—	+	—	E.c., Tet.	E.c., Tet.
Bennett[8]	++	NR	—	—	—	—	E.c.	E.c., Trich.
Obbard[5]	++	NR	—	—	—	—	E.c.	E.c.

TABLE X.—*continued.*

(B)—Acute Cases showing Amœbæ with included Red Blood Corpuscles.

Name	A	B	C	D	F	G	Other infections: Before end of emetin course	Other infections: After end of emetin course
Dorter[9]	+++	R	1	7	+	+	—	E.c.
Ball[6 11]	++	R	7	24	+	+	Tet., Trich.	Tet., Trich., T.c.
Barrie[6 11]	+++	R	9	5	+	+	Tet.	E.c., Tet.
Smith, C[9]	+++	R	1	7	+	+	—	E.c., Tet.
Rushforth[9]	++	R	1	12	—	—	E.c.	E.c., E.n., L.
Gaskin[10]	+	R	2	9	—	—	E.c.	E.c.

[1] When course of emetin injections was completed patient was given emetin ½ grain by the mouth for seven days.

[2] Was given later a twelve-day course of emetin one grain a day by the mouth to try and get rid of the *Entamœbæ coli* infection.

[3] Had a grain of emetin by the mouth on the two nights before the injection course was started.

[4] Had relapsed after eight injections of emetin three weeks before.

[5] Cured later by emetin one grain injection with ½ grain by the mouth each day for twelve days.

[6] Not treated further.

[7] Cured by course of emetin by mouth given immediately afterwards (5½ grains).

[8] Cured later by courses of one grain of emetin by mouth for twelve days.

[9] Relapsed again later after course of emetin by mouth with injections.

[10] Had relapses after emetin ½ grain a day for twelve days.

[11] Had resisted courses of emetin by mouth one grain a day for twelve days.

[12] Afterwards found resistant to 1½ grains of emetin by mouth for twelve days, ipecac. and thymol.

A = Degree or size of infection. B = Result. C = Number of days after commencement of treatment before infection disappeared. D = Number of days of control before cure or relapse was noted. F = History of previous dysentery. G = History of previous emetin treatment. CR = Cure. R = Relapse. NR = No reaction.

Two of the cases, Healy and Spiers, in Group I, were passing only cysts of *E. histolytica* and amœbæ without included red blood corpuscles when first seen, but both of these cases were just recovering from attacks of acute amœbic dysentery which they had had repeatedly for some years. They were really in a transition stage between the condition of the carrier and acute amœbic dysentery. In this group there were fifty-eight cases, of which six were acute cases of amœbic dysentery. Of the 52 carrier cases 37 were cured (CR), and did not relapse in the period of control, 10 relapsed (R), while in 5 the infection did not even disappear, there being apparently no reaction to the treatment (NR). The six acute cases all relapsed. It is interesting to note that of the carriers who relapsed only one gave a previous history of dysentery, while three of the five cases which did not react had had previous dysentery. Of the thirty-seven carriers which were cured fifteen

gave a history of dysentery. Two of the acute cases gave no history of dysentery, the attack in each case being a primary one.

The results are shown as follows:—

Carrier cases cured	37
,, ,, relapsed	10
,, ,, no reaction	5
Acute cases cured	0
,, ,, relapsed	6
,, ,, no reaction	0

Five of the carrier cases which relapsed, one of those which did not react to the emetin injections (Table X[5]) and one of the acute cases (Rushworth), were cured later by a combined course of emetin injections and emetin by the mouth (Group III); while another two of the carriers who did not react to the emetin injections were cured by a simple course of emetin by the mouth (Spiers and Bennett).

Group II.—Cases treated by a Twelve-day Course of One Grain of Emetin orally administered. Table XI.

As there had been a certain number of failures in the treatment by emetin injections in Group I it was decided to try a course of emetin by the mouth for twelve days. We were influenced in this direction by the remarkable result obtained in one case (Spiers) who had a long history of dysentery of three years, who had thirteen separate courses of emetin at one time or another and who had proved refractory to a course of emetin injections of one grain a day for twelve days (*see* Table X). This case with an enormous infection was given emetin by the mouth one grain a day for two days followed by $\frac{1}{2}$ grain a day for seven days. This treatment brought about an immediate and permanent cure (see history of case, Section I). In the series of cases treated in this way emetin was administered as a tinct. opii mixture as recommended by certain French physicians. The emetin, one grain, is dissolved in tinct. opii fifteen minims, and the fifteen-minim dose given in a cup of tea, preferably at night just before the patient goes to sleep. Having compared this method of administration with that of emetin in keratin-coated tabloids, we have found that it is much more difficult for the patients to retain the tea mixture without vomiting than the keratin-coated tabloids which we used extensively in a later series of cases. The latter, it is true, also often cause

vomiting, but this occurs later when the emetin tabloid has probably passed into the intestine.

In this group there were treated twelve cases, nine of which were carriers, and three acute amœbic dysenterics showing amœbæ with included red blood corpuscles. Of the nine carriers six were cured and half of these gave a history of previous dysentery. Two of the carriers relapsed and one did not react to the treatment. Of the three acute cases two relapsed and one did not react to treatment. The acute cases all had histories of previous dysentery and emetin treatment.

The results are as follow :—

Carrier cases cured	6
,, ,, relapsed	2
,, ,, no reaction	1
Acute cases cured	0
,, ,, relapsed	2
,, ,, no reaction	1

In this series one of the acute cases (Blair) received the emetin in keratin-coated tabloid instead of the tinct. opii tea mixture. This particular case (Blair) had been treated immediately before with methyl emetin sulphate two grains a day (one-grain injection and one grain by the mouth) for twelve days without any disappearance of the amœbæ.

In addition to the cases just mentioned, there were four others which were treated in a different manner, though still by means of emetin orally administered.

(1) Case Gaskin was given $\frac{1}{2}$ grain of emetin a day by the mouth in the tinct. opii tea mixture for twelve days. The case was one of acute amœbic dysentery showing amœbæ with included red blood corpuscles. There was no history of previous dysentery. The case relapsed nine days after treatment was completed.

(2) Case Spiers was given one grain of emetin in keratin-coated tabloids by the mouth each day for two days followed by half this dose for seven days. This course was commenced immediately on the completion of a twelve-day course of one-grain injections which had not caused a disappearance of the infection. In this case the cure was permanent.*

(3) Case Healy, who had failed to react to a twelve-day course of one-grain emetin injections, was given emetin $\frac{1}{2}$ grain a day in tinct. opii tea mixture for four days, followed by twice this dose for six days. This had no effect on the infection.

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* *See note on case, page* 181.

TABLE XI.—Cases treated by Emetin by the Mouth, One Grain a Day for Twelve Days.

(A) Carrier Cases not showing any Amœbæ with included Red Blood Corpuscles.

Name	A	B	C	D	E	F	G	Other infections before end of emetin course	Other infections after end of emetin course
Bennett[3] ..	++	CR	2	31	–	–	–	E.c.	E.c., Trich.
Duncan ..	+	CR	1	30	1	–	–	E.c., Tet.	E.c., Tet.
Lingard ..	+	CR	3	32	3	–	-	E.c., L., Tet.	E.c., L., Tet.
McGinty ..	++	CR	2	32	1	+	–	E.c., L.	E.c., L., Tet.
Howarth ..	+++	CR	2	36	–	+	+	–	E.c.
Dewhurst ..	++	CR	3	41	10	+	–	E.c.	E.c.
Jackson, A.[1] ..	++	R	10	3	4	–	–	Tet.	Tet.
Harris[2]	++	R	3	7	–	+	+	E.c., Trich.	Ec., Trich., Tet.
Main[1]	++	NR	–	–	1	–	–	Trich.	E.c., Trich.

(B) Acute Cases showing Amœbæ with included Red Blood Corpuscles.

Ball[1]	+++	R	1	11	1	+	+	Tet., Trich.	Tet., Trich.
Barrie[1] ..	++	NR	–	–	–	+	+	–	E.c., Tet.
Blair	+++	R	4	14	3	+	+	–	E.c.

Case treated by Emetin by the Mouth, ½ Grain a Day for Twelve Days.

Gaskin[1] ..	+	R	2	9	1	–	–	E.c.	E.c.

Case treated by Emetin by the Mouth, 1½ Grains for Four Days and One Grain for Eight Days.

Kettlewell[3] ..	+++	CR	3	32	2	+	+	E.c., Tet.	E.c., Tet.

Case treated by Emetin by the Mouth, One Grain for Two Days and ½ Grain for Seven Days.

Spiers[3]	+++	CR	1	32	2	+	+	–	–

Case treated by Emetin by Mouth, ½ Grain for Four Days and One Grain for Six Days.

Healy[3]	+++	NR	–	–	–	+	+	–	–

Case treated by Emetin, 1½ Grains by Mouth for Twelve Days.

Healy[4]	+++	R	4	3	1	+	+	–	–

[1] These cases were given later emetin injections, one grain for twelve days, and again relapsed.
[2] This case was given later emetin injections, one grain a day for twelve days, and was cured.
[3] These cases had already been given emetin injections, one grain a day for twelve days, without reaction immediately before this course.
[4] This case was receiving the second course of emetin by the mouth. Between the two courses there had been a complete course of ipecacuanha combined at the end with some emetin injections and a course of thymol, ten grains t.d.s., without result.

A = Degree or size of infection. B = Result. C = Number of days after commencement of treatment before infection disappeared. D = Number of days of control before cure of relapse was noted. E = Number of occasions patient vomited after emetin. F = History of previous dysentery. G = History of previous emetin treatment. CR = Cure. R = Relapse. NR = No reaction.

(4) Case Healy again was given emetin by the mouth after an interval during which he was treated with ipecacuanha and thymol. On this occasion, he was placed on very strict dysentery diet and given by the mouth 1½ grains of emetin a day for twelve days. The infection disappeared, but returned in three days after the course was completed.

(5) Case Kettlewell, who had not reacted to a twelve-day course of one-grain emetin injections, was given emetin by the mouth in a dose of 1½ grains a day for four days followed by one grain a day for eight days. During the treatment the patient was kept in bed on dysentery diet. The infection disappeared and did not recur.

Group III.—Cases treated by Emetin administered both hypodermically and orally (1½ *Grains a day for Twelve Days*). *Table XII.*

The cases in this group were all treated on a combined oral and injection therapy. They were kept strictly in bed and given milk or beef tea diet and received each morning during twelve days a one-grain emetin injection and each evening ½ grain of emetin in keratin-coated tabloid by the mouth. The vomiting in these cases was, as a rule, less frequent than when the emetin was given in the tinct. opii tea mixture, and when it occurred it was generally an hour or more after the tabloid had been taken. In this manner were treated thirty-eight cases, of which thirty were simple carriers without dysentery, and eight were acute cases of amœbic dysentery showing active amœbæ with included red blood corpuscles. It will be seen that a cure was obtained in all the carrier cases, though six of these (Table X[5]) had previously relapsed after a course of emetin injections of one grain a day for twelve days. Of the eight acute cases there were five relapses and two cures, while one case (Jackson) was not sufficiently controlled after treatment (only nine days). Two of the acute cases which relapsed (Dorter and Smith) and one which was cured (Rushforth) had already relapsed after treatment by emetin injections of one grain a day for twelve days (Table X). The results are as follow:—

Carrier cases cured	30
,, ,, relapsed	0
,, ,, no reaction	0
Acute cases cured	2
,, ,, relapsed	5
,, ,, no reaction	0

TABLE XII. — Cases treated by 1½ Grains of Emetin for Twelve Days. One-grain Injection each Morning and ½ Grain in Keratin-coated Tabloid by the Mouth each Night.

(A) Carrier Cases not showing any Amœbæ with included Red Blood Corpuscles.

Name	A	B	C	D	E	F	G	H	Other Infections: Before end of emetin course	Other Infections: After end of emetin course
Weber[1]	++	CR	2	40	1	+	+	10	E.c., Coc.	E.c., E.n.
Jordan	+	CR	2	28	–	+	–	–	E.c., L., E.n.	E.c., L.
Morgan, H. ..	+++	?	10	9	2	+	+	–	Tet.	Tet.
Lyall	+++	CR	2	41	–	–	–	–	E.n.	–
Borg	+	CR	3	33	–	–	–	–	E.c., E.n.	E.c.
Palmer	++	CR	1	34	–	–	–	–	E.c., L., Tet.	E.c., Tet.
English.. ..	++	CR	1	33	6	–	–	–	E.c., E.n.	E.n.
Howard.. ..	+	CR	1	31	–	–	–	–	E.c., Tet., Trich.	E.c.. Tet. Trich.
Taylor	+	CR	5	34	1	–	–	–	L.	–
Hyde	++	CR	1	40	4	–	–	–	E.n., Trich.	E.n., Tet., Trich.
Eastdown ..	+++	CR	2	34	–	–	–	–	E.c.	E.c.
Cherril	+++	CR	3	32	1	–	–	–	E.c., E.n., L.	E.c., E.n.
Liddle	+++	CR	1	35	–	–	–	–	E.c., E.n., Trich.	–
Morgan, B. ..	++	CR	1	51	7	–	–	–	–	–
Wing	+	CR	?	47	1	–	–	–	E.c., E.n., L.	E.c., E.n.,L., Tet.
Sargeant ..	+	CR	3	32	2	–	–	–	E.c., E.n., L., Tet.	E.c., E.n., Tet.
Baker	++	CR	?	32	1	–	–	–	–	E.c., L.
Graham.. ..	+++	CR	3	32	–	–	–	–	E.c., E.n.	E.n., Tet.
Reedie	+	CR	2	35	6	–	–	–	T.c.	–
Ross	+	CR	1	31	–	–	–	–	T.c.	–
Obbard[1] ..	++	CR	1	48	3	–	–	10	E.c.	E.c.
Pero[1 2]	+++	CR	2	30	6	–	–	10	Tet.	Tet.
McQuade[1] ..	+++	CR	1	31	–	–	–	26	Tet.	–
Squires[1].. ..	++	CR	2	32	–	–	–	16	E.c., E.n. T.c.	E.c., En.
Boyd[1]	++	CR	3	32	–	–	–	17	E.c., Tet., T.c.	E.c., Tet.
Carr	+++	CR	1	36	–	–	–	–	E.n.	–
Miller	+++	CR	3	36	1	–	–	–	–	–
Downs	+++	CR	2	36	–	–	–	–	E.c.	E.c.
Triphook ..	+	CR	2	34	–	–	–	–	–	–
Lloyd	+++	CR	5	29	2	–	–	–	E.c.	E.c., L.

(B) Acute Cases showing Active Amœbæ with included Red Blood Corpuscles.

Hollow	+++	CR	2	36	1	+	+	–	Trich.	Trich.
Rushforth[1] ..	++	CR	2	57	1	–	–	18	E.c., E.n.	E.c., E.n.
Russell, H. ..	+++	R	1	3	–	+	+	–	–	–
Dorter[1]	++	R	1	11	–	+	+	9	E.c.	E.c.
Smith[1]	+++	R	–	6	–	+	+	34	E.c., Tet.	Tet.
Greenwood ..	+++	R	12	14	1	+	–	–	–	–
Jackson	+++	?	2	9	1	+	–	–	L.	L.
Wilkinson ..	+	R	–	23	1	–	–	–	E.c., Tet.	E.c., Tet.

[1] These cases had relapsed after or failed to react to a previous twelve-day course of emetin (one grain a day injection).

[2] Owing to persistent vomiting the ½-grain tabloid of emetin was not given after the sixth day.

A = Degree or size of infection. B = Result. C = Number of days after commencement of treatment before infection disappeared. D = Number of days of control before cure or relapse was noted. E = Number of occasions patient vomited after emetin. F = History of previous dysentery. G = History of previous emetin treatment. H = Interval in days between the termination of a previous course of emetin (one grain a day injection) and the commencement of the present course, both courses during the present observation. CR = Cure. R = Relapse. NR = No reaction.

(*b*) *Treatment by Methyl Emetin Sulphate.*—Methyl emetin in the form of the sulphate was tried on four cases. This drug prepared by Dr. Pyman of the Wellcome Bureau of Scientific Research is a stable compound which is soluble in water and not decomposed by boiling. As methyl emetin, according to experiments made by one of us (C. M. W. 1915), is equal in amœbicidal power to the emetin hydrochloride, and as it is much less toxic to animals on injection, it was decided to give the drug a trial in amœbic affections in man. Dr. Low had previously tried another salt of methyl emetin on a case of rather doubtful nature at the Albert Dock Hospital, but the investigation was not carried very far and no attempt was made to test its action in comparison with the usually employed emetin hydrochloride.

We have tested methyl emetin sulphate on four cases. Three of these were acute cases of amœbic dysentery, while one was a carrier. The carrier case (Percival) had a +++ infection of *E. histolytica* and a smaller *E. coli* infection. The patient was given for twelve days an injection of the drug each morning (one grain in one cubic centimetre of distilled water) and one grain in keratin-coated tabloids by the mouth each night. The *E. histolytica* infection disappeared after the seventh day of treatment and did not recur during a subsequent control of over one month. During the treatment the patient was kept in bed on milk diet.

Of the acute cases one (Smith)* had already relapsed after two courses of emetin hydrochloride (Tables X and XII). He was given the same treatment as the preceding case. The course of methyl emetin apparently had no action on the infection, for free forms of *E. histolytica*, sometimes with included red blood corpuscles, were passed during the whole course. The second acute case treated was Russell—who had also relapsed after a course of emetin hydrochloride ($1\frac{1}{2}$ grains a day subcutaneously and orally: Table XII). The same treatment with methyl emetin sulphate as was used in the other cases was adopted. On this occasion the infection disappeared after the fourth day of treatment, but reappeared three days after the completion of the course. The third acute case (Blair) was given the same dose, but it did not even bring about a disappearance of the infection. Accordingly at the end of the twelve-day course of methyl emetin sulphate the treatment was changed to one grain of emetin hydrochloride in keratin-coated tabloid at night. This alteration of the emetin was made without the patient's knowledge. The result

* *See note on case, page* 187.

was that for the first three nights vomiting occurred within a short time of taking the drug, though there had been no vomiting previously. This illustrates very clearly the difference as regards the property of producing nausea and vomiting between the two drugs, methyl emetin sulphate and emetin hydrochloride.

From the results it seems clear that with acute cases the methyl emetin in the dose employed has not the same power of ridding an individual of infection as the emetin hydrochloride. In two of the cases the infection did not even disappear, while in the third it vanished only to return soon after the completion of the course. It must be remembered that the dose employed (two grains a day) was larger than the largest dose of emetin hydrochloride used by us ($1\frac{1}{2}$ grains a day). The single carrier case, however, cleared up, and no relapse occurred, so that it is clear the drug has an action on the parasite but is not as potent as the usually employed hydrochloride of emetin. It is possible that in larger doses a better result could be obtained. An important feature of the drug is that the nausea and vomiting which so often follow the oral administration of emetin hydrochloride are absent. The patients who had taken the emetin hydrochloride by mouth and who had experienced the nausea following it, stated that they could go on taking the methyl emetin quite easily, and this when double the dose was being given.

It may be stated safely, therefore, that methyl emetin sulphate has not such a marked action on *E. histolytica* as emetin hydrochloride, but that it causes much less vomiting and feeling of nausea than the latter drug.

(*c*) *Treatment by Thymol.*—Owing to a view which had been expressed that thymol as an intestinal disinfectant was a very powerful agent this drug was tried in one case. This was Healy, who had a very large infection of *E. histolytica* (cysts and free forms), and who had resisted a course of one-grain emetin injections, a course of emetin by the mouth and a course of pulv. ipecac. Thymol, ten grains three times a day, was given, but it was absolutely without action on the infection.

(*d*) *Treatment by Pulv. Ipecac.*—The same case, Healy, after having resisted the course of emetin injections and emetin orally, was placed on a course of pulv. ipecac. He was given to start with thirty grains a day, and this was reduced by five grains daily, till a dose of ten grains was reached. This dose was then kept up for eight days, and for the last three days an injection of $\frac{1}{2}$ grain of emetin was given also. The treatment merely had the effect of temporarily diminishing the infection.

General Considerations as regards Treatment.

(*a*) *Is the Object of Treatment to destroy the Cysts or the Amœbæ or both ?*—In the treatment of cases of *E. histolytica* infections there is often supposed to be a difference between the carrier case passing cysts and amœbæ in a comparatively normal stool and the acute case passing only amœbæ in the dysenteric stool. The idea has arisen from the notion that the cysts are more resistant than the unprotected amœbæ. It is a fact that the cysts are more resistant, but this has really no bearing on the question of the treatment. As has been explained above there is no real difference between the carrier and the acute case, the two conditions being merely different stages of the same disease. The cysts of *E. histolytica* are formed by amœbæ which are living about the ulcers in the large intestine, and when once a cyst has been produced it has no more influence on the course of the infection. A cyst cannot rupture and liberate its amœbæ in the large intestine. For this to happen it has to find its way to the small intestine where it can come under the action of the pancreatic fluid, and this it cannot do unless it escapes from the intestine and is ingested by another or the same host. This being the case it would probably be a very good thing for an infected individual if all the amœbæ in the intestine could be induced to encyst, for they would then have no course open to them but to pass out in the fæces.

In treating cases of *E. histolytica* infection it is, fortunately, not the resistant cysts we have to deal with, but the free amœbæ which must be present in the gut if the cysts are found in the stool. The cysts in fact are very useful guides as an indication of the extent of the amœbic infection of the gut in just the same way as the number of ankylostoma eggs is an indication of the number of worms present in the small intestine. In treating an ankylostome infection one does not endeavour to destroy the eggs in the intestine any more than in treating an *E. histolytica* infection one tries to destroy the cysts. In both cases, the eggs and the cysts are conveniently searched for in the stool to estimate the effect of any course of treatment aimed at the destruction of the worms and the amœbæ respectively. We have dwelt upon this point because we have so often heard it stated that some cases of *E. histolytica* infection will not respond to treatment, merely because the resistant cysts are present in the gut. This view is entirely disproved from the results we have obtained by emetin treatment, and one of these cases (Smith) who relapsed repeatedly never passed cysts at any

time during a long observation. The destruction of the cysts is an important matter after they have left the body, for if they are not destroyed they are likely to be carried about by water and flies and give rise to infection in other people. For this reason the stools of dysentery carriers should be disinfected with cresol 1 in 10, and care should be taken that the cysts are not disseminated on the hands or in any other way.

(*b*) *Comparison of the Different Methods of Emetin Treatment.*—The results obtained in the three groups of cases described above can be tabulated as follow. In each group the course of treatment extended over twelve days :—

	Emetin one grain a day injection	Emetin one grain a day by the mouth	Emetin one grain a day injection; ½ grain a day by the mouth
Carrier cases cured ..	37 ..	6 ..	30
,, ,, relapsed ..	10 ..	2 ..	0
,, ,, no reaction	5 ..	1 ..	0
Acute cases cured ..	0 ..	0 ..	2
,, ,, relapsed ..	6 ..	2 ..	5
,, ,, no reaction ..	0 ..	1 ..	0

As regards the first two groups the results with the carriers are almost identical, as can be seen by multiplying the smaller figures in Group II by six. There is, however, a slight balance in favour of the one-grain injection over the one grain by the mouth. As regards the acute cases it will be seen that all the cases relapsed, though the balance is again in favour of the injection method of treatment, as one of the cases treated by the mouth showed no reaction.

When we come to the combined treatment (emetin injections one grain a day and emetin by the mouth ½ grain a day) the results are distinctly better both with the carriers and acute cases. With the former there were treated thirty cases and none of those relapsed during the period of control. With the acute cases two were cured and four relapsed. In addition to the combined injection and oral method of emetin administration these cases were kept in bed on milk or beef-tea diet, and this may have had something to do with the better results, for the cases treated under Groups I and II were not kept in bed and had light chicken diet. The acute cases, however, were all kept in bed on dysentery diet. On the other hand, the cases treated in Group I were given sodium sulphate mixture several times a day, while in Groups II and III the cases were not given the mixture regularly as the emetin administered by the mouth tended to keep the bowels loose, so that the saline mixture was usually unnecessary.

It is important to note that five of the carrier cases which relapsed after treatment in Group I were treated later and cured as in Group III, while one carrier case which did not react at all to the treatment as in Group I was cured later by treatment as in Group II, while case Spiers who resisted the one grain a day by injections was cured by a course of emetin by the mouth immediately after.

It seems to follow from this that certain cases that prove refractory to one line of treatment or one method of emetin administration may respond to emetin administered in another way. It is possibly this factor which has given the better results with the combined method of treatment.

(*c*) *Carrier and Acute Cases.*—It will have been seen from the list given on *page* 110 that the ordinary carrier cases not showing any or only slight symptoms are much more readily cured than the acute cases. Thus all the six acute cases in Group I relapsed after emetin injections of one grain a day for twelve days. In Group II again, where three acute cases were treated by emetin by the mouth (one grain a day), one case did not even react to the treatment, while two relapsed. In the third group there were treated seven acute cases, and of these five relapsed and two were cured.

It is quite evident, therefore, that the cases which are most difficult to rid of infection are not the carrier cases passing cysts without any symptoms, but the acute cases passing blood and mucus in the stool with active amœbæ containing red blood corpuscles. The acute cases have nearly all had a history of recurrent attacks of dysentery and generally previous emetin treatment. Three of these cases, however, were treated at their first attack of dysentery (Rushworth, Wilkinson and Gaskin), yet all of them relapsed. Rushworth, however, was subsequently cured by a course of emetin given by injection and by the mouth (Table XII).

The action of emetin on the carrier case generally causes the disappearance of cysts from the stool in two or three days, and similarly in the acute cases not only do the amœbæ disappear very quickly but the symptoms clear up also and the patient to all intents and purposes has recovered from his dysentery. It is this fact which had led to the idea that emetin in small doses is such a sure specific for amœbic dysentery. It is only by the detailed control of the cases, as in the methods adopted by us in these observations, that the subsequent course of the cases can be followed. It has resulted that in almost every instance relapse has taken place.

The acute cases seem to differ from the carrier cases only in degree, for between the attacks of dysentery the acute cases are actually in the carrier condition passing amœbæ and cysts which are indistinguishable from those passed by the carriers who have, perhaps, never had an attack of dysentery. The view which has already been expressed above, that it seems probable that all the carriers have some ulceration of the intestine, affords the best explanation of this difference in the action of emetin in the acute and carrier cases. Certain of the carrier cases have no symptoms whatever and it is possible that in them the ulceration is at a minimum. Other carriers have had an attack of dysentery and from time to time pass mucus in the stool. In them the ulceration is probably more extensive. With a still more serious condition of ulceration the attacks of dysentery are more frequent and we get the condition of the typical chronic amœbic dysenteric who has repeated attacks of dysentery. During these he passes blood and mucus with active amœbæ containing red blood cells. Between the attacks the stool is always soft, and contains varying quantities of mucus, while *E. histolytica* cysts and amœbæ are found in enormous numbers. The most serious condition is seen in cases like that of Smith where the dysentery is practically constant and encysted forms of *E. histolytica* never occur in the stool. The amœbæ are always in the free condition and frequently show included red blood corpuscles. These cases with extensive ulceration are naturally much more difficult to treat than the simple carriers who have only very slight intestinal lesions. It seems probable that the varying condition of the gut rather than any other factor is responsible for the variation in the response to emetin treatment. We do not know how the emetin reaches the amœbæ or exactly how it is excreted, for cures have been effected both by hypodermic injection of emetin as well as by emetin by the mouth, but if the drug is usually brought into contact with the amœbæ by way of the circulation then it is perhaps understandable that, in cases with the extensive ulceration of the chronic amœbic dysenteric, with old fibrotic ulcers and thrombosed vessels, the emetin is unable to reach many of the amœbæ for purely mechanical reasons of impaired circulation. It is possible that in this condition the amœbæ in certain situations can be reached and destroyed if emetin is given by the mouth instead of by injection.

(*d*) *Are there Emetin-resistant Strains of Amœbæ* ?—It has been suggested that, in certain cases, the amœbæ owing to past inefficient emetin treatment have acquired the power of resisting the

drug. It is very difficult to obtain precise information on this point, for, as already explained, the failure of emetin to cure may be due to purely mechanical reasons of poor circulation or other causes. Case Spiers is of much interest in this connection. There was a long history of dysentery of three years standing, and there had been in all thirteen courses of emetin, after each of which the patient's symptoms disappeared. The patient himself had also been in the habit of taking one or two ½-grain emetin tabloids for this purpose. Such a case had every chance of developing a resistant strain of amœbæ, and when a twelve-day course of one-grain injections failed entirely to influence the infection we were under the impression that the strain of *E. histolytica* was resistant to emetin. This view was entirely disproved by the fact that a course of emetin by the mouth over nine days (5½ grains in all) completely got rid of the infection without any relapse taking place. In this case at any rate it seemed as if the emetin administered by the mouth reached the amœbæ more readily than the emetin by injections. Again, case Healy,* who had a still longer history of chronic amœbic dysentery, failed entirely to react to emetin injections. He failed also to react to small doses of emetin by the mouth and also to a course of treatment by pulv. ipecac. Yet a course of emetin by the mouth 1½ grains a day caused the infection to disappear at least for some days.

It, therefore, seems to us that because an infection does not appear to respond to treatment by emetin injections it does not follow that this failure is due to the presence of a resistant strain, for if the emetin can be made to reach the amœbæ by some other method of administration in most cases some response or even cure will be the result.

The same failure to respond to emetin injections is seen sometimes in the case of amœbic abscess of the liver. A case of this kind was seen by one of us (C. M. W.) in London last year, where a draining abscess of the liver continued to discharge active amœbæ though a complete course of emetin injections of a grain a day for twelve days was given. A second case of this kind came under our notice a few months ago. We were controlling a case by examinations of the fæces and liver abscess pus for Captain Redmond, R.A.M.C., who was treating the case. The fæces showed no sign of amœbic infection, while the pus from the abscess contained constantly numbers of *E. histolytica*, and this in spite of the fact that the patient received a full course of emetin injections

See note on page 181.

for twelve days, a course of emetin by the mouth, and in addition, irrigation of the abscess cavity with a solution of emetin. The amœbæ were very active and included many red blood corpuscles, an observation which at once disproves the view expressed by Escomel (*Bull. Soc. Path. Exot.*, October, 1913), that *E. histolytica* will not ingest the red blood corpuscles of a person receiving emetin injections. On one occasion a small quantity of the pus was mixed with a sclution of emetin hydrochloride (one grain in one cubic centimetre, or 6·5 per cent. solution), and a preparation of the mixture watched under the microscope. The amœbæ, instead of being killed at once, moved about actively in the liquid and only came finally to rest after the expiry of ten minutes. We cannot state the exact strength of the emetin solution in which the amœbæ were moving, but it was many times in excess of the usual 1 in 100,000, which is claimed to kill them at once. Whether these amœbæ were emetin-resistant or not cannot be decided till similar observations are made with amœbæ from abscesses in cases which have not received previous emetin treatment. The case just recorded recovered in the usual way by drainage of the abscess cavity, but there was no evidence to show that the various courses of emetin had any influence in bringing about this fortunate result. It has been claimed that emetin injections will not only clear up the presuppurative hepatitis of amœbic abscess, but it will sometimes cause an actual unopened abscess to disappear. The case recorded above does not offer any explanation of this, though it is possible that the chances of the emetin reaching the amœbæ in an abscess wall vary just as they do in the gut. Some abscesses may have a good circulation in the surrounding tissues, so that the emetin can reach the amœbæ ; while in other abscesses, probably of a long duration, the circulation is poor owing to the occlusion of the blood-vessels by thrombosis, fibrosis or other obstruction.

(*e*) *Does Emetin tend to make the Amœbæ encyst?*—It has been stated that the effect of emetin, especially in inadaquate doses, is to make the *E. histolytica* encyst and to convert an acute into a carrier case. As a rule a protozoan encysts either because it wishes to protect itself against some adverse influence or for reproductive purposes, so the presence of cysts of *E. histolytica* is looked upon as an indication of some adverse influence acting upon the amœbæ in the gut. It was thought, therefore, that if emetin was given in insufficient doses to kill the amœbæ they would tend to protect themselves by encysting. The encystment of the *E. histolytica* in the intestine is not such a simple matter as might

at first sight appear. A case with acute dysentery is passing large entamœbæ quite unlike the encysting generation of small amœbæ, and if emetin is to cause the amœbæ to encyst we must suppose that its first effect is to make the large amœbæ divide rapidly to produce small forms, and that these must take on the characters of the pre-encysting "minuta" forms of *E. histolytica*. This is quite another matter than the supposed quick secretion of a capsule round an amœba, because it is being irritated by small doses of emetin.

We have already seen that the natural course of an amœbic infection is that of the carrier case, and that in a certain percentage of these where the ulceration of the gut is extensive attacks of dysentery manifest themselves from time to time, while, between the attacks, the condition of the carrier is reverted to. In whatever way a person becomes infected with *E. histolytica*, whether by an initial attack of amœbic dysentery or not, the natural tendency is towards the development of the carrier condition. This being the case it is manifestly impossible to regard inadequate emetin treatment as a factor which increases the number of carrier cases, for these cases if left untreated would have become carriers in any case. Moreover, the supposed inadequate treatment would probably have cured a few cases at least, so must have reduced the total number of possible carriers.

It is quite possible that when the "encysting" or "minuta" generation of *E. histolytica* is present in the intestine some adverse influence might suddenly cause an encystment of numbers of these, but before we can decide this point we must understand the normal course of such infections, judged by the appearance in the stools. A person who is in the carrier condition passes both cysts of *E. histolytica* and minuta forms of amœbæ, but these are not passed regularly. On some days cysts alone are passed, while on others the amœbæ are most numerous or alone present. Much depends on the consistency of the stool. In some of the carrier cases treated by us the emetin course was commenced only when cysts were being passed and the immediate effect has appeared to be the disappearance of the cyst with the passage of free amœbæ. On the other hand the reverse has occurred, while generally in the cases of pure *E. histolytica* infection the cysts have tended to disappear from the stool before the amœbæ. The results we have obtained do not afford any evidence in favour of the view that the emetin causes the amœbæ in the gut to encyst. Nor have we been able to obtain any evidence that the emetin treatment causes any particular form of cyst to appear in the stool. In the examinations for carrier

cases we have noted that, though most usually the infections show a majority of cysts with four nuclei, with a smaller number of one and two-nuclear cysts, at times infections are encountered where nearly every cyst present has only one nucleus. This has appeared also in our control of individual cases in hospital, and it seems evident that the forms which actually appear in the stool are dependent on the rate of emptying of the large intestine rather than on any peculiar action treatment may be having on the amœbæ which are there.

(*f*) *Influence of other Infections on the Treatment.*—A glance at the tables of the three main groups of cases treated shows that the majority of cases had some other protozoal infection besides the *E. histolytica*. These coincident infections have been arranged in two columns according as to whether they were discovered before or after the completion of the course of emetin treatment. Many of these only appeared and were detected after several days' observation of the case. Without entering into any details it seems quite clear from the tables that amongst the carriers which were cured there were just as many extra infections, which were just as varied as amongst the carriers which relapsed, so that the presence of flagellates or *E. coli* did not in any way prejudice the case against a successful emetin treatment. Amongst the acute cases again extra infections were the rule, but as practically all the cases relapsed it is not possible to judge if the result was in any way due to their presence. Generally speaking our results seem to indicate that the action of emetin on *E. histolytica* is not affected by the presence of other protozoa, and we can find no evidence in support of the view expressed by Dr. Barlow (*New York Med. Journ.*, 1915), that the cases most difficult to treat are those complicated by flagellate infections. In cases treated with emetin by the mouth it was the rule for all the coincident infections to disappear, but in nearly every instance they reappeared later during the period of control.

If one looks at the protocols of the cases treated by the combined method of orally and subcutaneously administered emetin it will be seen how all the infections disappear soon after treatment is commenced. This is illustrated by the occurrence of a complete blank on the charts corresponding to the course of emetin. After the course is completed the columns begin to fill up as the infections reappear.

(*g*) *Influence of Diet and Rest in Bed on the Treatment.*—It has already been explained that the carrier cases appearing in

Tables X and XI were given a chicken diet, and were not kept in bed during treatment. The acute cases in these two groups were, however, given dysentery diet and kept in bed. It is difficult to state whether there would have been fewer relapses amongst the carriers in these groups if rest in bed and special milk diet or dysentery diet had been enforced. Rest in bed and milk diet were ordered for the cases appearing in Table XII, and here all the carrier cases were cured. The difference in the result, however, is more probably due to the extra dose of emetin which the latter received. That very bad cases can be cured without any rest in bed is well illustrated by case Spiers, who had, as already mentioned, a very bad history of dysentery and repeated emetin treatments. He was treated whilst he still carried on his usual office occupation, and confined himself to an ordinary light chicken diet. He received during twelve days an injection of a grain of emetin a day, which did not rid him of the infection. This was followed by a course of emetin ($5\frac{1}{2}$ grains) by the mouth which permanently cured the patient. This very satisfactory result was obtained without any special rest or diet. Two other carriers (Cox and Badham) were treated by twelve one-grain emetin injections, while they continued on duty and took full diet. One of these was cured and the other relapsed. Another case (Ball) had already had an attack of acute amœbic dysentery, for which he had been treated in bed by oral administration of emetin. He relapsed into the carrier condition and was then treated while still on duty by twelve daily one-grain emetin injections. He relapsed again after the second course. Thus, of the four cases treated out of hospital two were cured and two relapsed. It does not seem advisable, however, to treat patients with emetin while still on duty, though this may have to be done in special cases.

When patients are in hospital, and especially when they are in bed, they do not need a full diet, and for this reason it is sufficient to give them milk or other light diet alone. When emetin is being taken by the mouth, and possibly comes into contact with the amœbæ by way of the intestinal tract, an overloaded intestine would be a disadvantage in that the available emetin would be reduced. Furthermore, the tendency to vomit would be greater if too much food were taken. This, of course, does not apply to emetin administered by injection only, as this seems to produce very little, if any, tendency to vomiting. Case Healy, who had proved most refractory to treatment and who had had twelve one-grain injections, a course of emetin (eight grains) by the mouth

and a full course of treatment by pulv. ipecac. without his infection disappearing, was finally treated on what was almost a starvation diet. He was kept in bed and given the diet of an acute dysenteric (barley water, arrowroot water, jelly, tea with a little milk and custard). This diet was started two days before a course of emetin ($1\frac{1}{2}$ grains a day) by the mouth was commenced, and was continued during the twelve-day course of treatment. The infection disappeared, but three days later there were present as many cysts and amœbæ as before. A second case, Kettlewell, who had resisted the twelve one-grain injections, was treated in a similar manner, but with less emetin (fourteen grains during twelve days). The result in this case was a cure without subsequent relapse. It seems, however, that in most cases where there are no actual dysenteric symptoms such a rigid diet is hardly necessary and may possibly do harm in weakening the patient and rendering him more susceptible to the possible action of emetin on the heart. Case Kettlewell was one of the two cases mentioned below in which some irregularity in the heart's action was noted after treatment. In most instances for carrier cases in bed a milk diet with eggs, bread and butter and milk puddings is quite suitable. Those with actual dysentery with blood and mucus in the stool may require a more rigid diet.

(*h*) *Influence of Salines on the Treatment.*—All the cases in Group I were given a mixture containing sodium sulphate one dram four or more times a day, with a view to flushing out the gut. The cases in the second group were not given the mixture so regularly, while in the third group they seldom had it. As already mentioned, emetin by the mouth itself tends to keep the bowels loose, so that salines are not needed so frequently for this purpose. With the acute cases the bowels are generally quite loose without any salines. From the cases we have treated it does not seem that the salines have influenced the result in any way. In fact, the regular administration of salines does not tend to produce such a constant washing out of the large intestine as is often supposed. We have noted that patients who take sodium sulphate mixture regularly so many times a day may pass watery stools for the first two days, but that the effect of the saline seems to pass off. A better result would probably be obtained by giving a single large dose of saline each morning. Barlow (*New York Med. Journ.*, October, 1915) has recently expressed the opinion that emetin has a better chance of action on the amœbæ if there is not too much flushing of the gut, and he advocates the administration of a saline every five or six days only to clear out the large intestine.

(*i*) *Effect of Emetin on the Patient. Gastro-Intestinal.*—Emetin administered by the mouth causes vomiting in many instances. In the Tables XI and XII, showing the various groups of cases treated, the number of times each patient vomited, if at all, is shown in a special column (E). This only occurred after emetin orally administered, and the patients did not vomit more than once after any single dose. Emetin injections do not produce vomiting, though some complain of a feeling of nausea. At first the drug was given, as explained above, dissolved in tinct. opii, the requisite dose being taken in a cup of tea at night. This caused vomiting very easily, especially in some cases, so that finally this method was abandoned in favour of emetin in the form of keratin-coated tabloids. Even this caused a good deal of vomiting, but usually it was an hour or more after taking the dose. In the group of cases treated both by injection and orally, one case (Pero) vomited almost at once after each tabloid, so that after six nights the ½-grain tabloid by the mouth had to be discontinued. Vomiting so quickly after the tabloid was, however, the exception. One of the cases (Dewhurst), treated exclusively by emetin by the mouth in the tinct. opii tea mixture, vomited after ten of the twelve doses, as will be seen by the chart of his case. In spite of this the *E. histolytica* infection disappeared and there was no recurrence. Case Spiers, who had resisted twelve injections of emetin, was given emetin by the mouth, one grain in keratin-coated capsule. The dose was taken at night before going to sleep, but though the patient made every effort to prevent this, he vomited on two nights, once after two hours and once after three. Nevertheless, the amœbæ disappeared immediately from the stool, though they were present in enormous numbers before. It is thus evident that though the drug may cause vomiting the bulk of the dose taken is retained. As regards the vomiting after the drug, a great deal depends upon the determination of the patient to withstand the inclination. When several patients in one ward are receiving the treatment it often happens that if one of them vomits the others follow suit at once. One case (Healy), which proved very resistant to emetin, was given emetin 1½ grains by the mouth for twelve days. There was a strong feeling of nausea, but the patient made heroic efforts and was able to prevent himself from vomiting on every occasion except after the first dose, though this was not till six hours after it was taken.

It seems that a certain resistance to the nauseating effect can be acquired, and though the first two or three doses may be followed

by a desire to vomit, the later doses can be taken with impunity. Case Healy, who took the 1½ grains for twelve days, found this most marked, and felt that finally he could have continued without any ill effect in this direction.

Emetin administered by the mouth, as already explained, tends to produce looseness of the bowels, so that if the drug is being administered in this way it is unnecessary to give salines. We have not noted any tendency to diarrhœa as a result of emetin administered subcutaneously alone, nor has the emetin produced any hæmorrhage from the bowel, as it is supposed to do sometimes by causing some change in the vessels about the ulcerated areas.

Local.—The subcutaneous injection of emetin has produced very little local effect. The only cases which have complained of any local trouble have been nervous, highly sensitive individuals who have stated that the injection of emetin was followed by pain at the site of injection. The majority of cases, however, have taken the injections without any complaint. In one case, in which twelve one-grain injections were given into the right arm, the last injection, the only one given in the forearm, produced a tingling in the hand and elbow. It is probable that in this case a nerve was accidentally injured.

General.—In certain cases the emetin seems to produce a feeling of stiffness and weakness in all the limbs and a general lassitude. Case Spiers, who had the twelve-grain course of emetin while he carried on his regular office work, noted this weakness especially in the legs.

Heart.—The possible deleterious action of emetin on the heart is of great importance. It has been reported from time to time that emetin has caused not only derangement of the heart's action but even death in some instances. Dale has noted that in cats there is definite evidence that emetin in large doses is cumulative in its action, so that care has to be exercised in its administration. On this account we have paid very special attention to this point in the treatment of our cases, the pulse-rate and temperature having been kept regularly. We can say that in only two instances were any signs of heart trouble noted. In one case (Kettlewell) there had been given twelve one-grain injections of emetin, and as this had not abolished the infection the patient was placed on dysentery diet, kept in bed and given 1½ grains of emetin by the mouth for four days, followed by one grain for eight days. When he got up after the treatment he felt weak, a condition which must have resulted not only from the emetin he had had, but also from

the low diet and stay in bed. He suffered from shortness of breath on exertion, and when he was discharged to a convalescent home this became more marked and was accompanied by irregularity in the action of the heart. The patient was kept at rest as much as possible and in the course of three weeks the condition passed off, when he returned to full duty. Another case which did not react to twelve one-grain injections of emetin was then treated by twelve one-grain injections with $\frac{1}{2}$ grain by the mouth. The patient was kept in bed on a milk diet. Shortness of breath and irregularity of the heart were noted when he got up after treatment. The condition passed off in a couple of weeks.

In none of the other cases treated was anything comparable noted. It seems evident that certain individuals are more easily affected by the drug than others, and it is probable that such have an idiosyncrasy to the drug just as certain individuals have to quinine. In the great majority of cases, however, emetin produces no such symptoms, and case Healy, who had in all forty-six grains of emetin together with a course of pulv. ipecac. during a period of fifty-three days, was quite immune to the drug from this point of view.

In order, therefore, to be on the safe side it is better to keep the patient in bed, especially when more than one grain of emetin a day is being administered and when the patient is being kept on the low diet, for this in itself tends to reduce the resisting powers. Furthermore, a patient in bed is under better control as regards his diet, and it is easier to make observations on the temperature and pulse under these conditions. The collection of samples of the stool for examination is also facilitated.

Temperature.—In none of the cases treated did the emetin appear to have any effect on the temperature. In those in which some irregularity was noted during the course of emetin some other factor was present. In one case peculiar unexplained elevations of temperature were found to be due to appendicitis, and in another case to malaria.

(*j*) *When does the Infection disappear under Emetin Treatment?*—In Tables X, XI and XII (Columns C) are inserted showing the number of days before infections disappeared after emetin was commenced. In the cases in which there was no reaction (NR) to the emetin treatment there was no disappearance of infection, accordingly a blank is left. In other cases where a ? is inserted the exact day of disappearance of infection is doubtful, either because the infection was temporarily absent at the commencement

of treatment or because only free amœbæ were present, which could not be distinguished from *E. coli* which were present also.

Of thirty-five carrier cases which received one grain of emetin subcutaneously for twelve days and which did not relapse, the infection persisted on an average for four days before disappearing, while of ten carrier cases which were treated in the same way and which relapsed later, the figure is 4·5 days. On the other hand, for twenty-seven carrier cases treated and cured by the combined subcutaneous and oral method (1½ grains a day) the average is only two days. Amongst the carriers treated in this way there were no relapses. The cases which were treated by one grain of emetin by the mouth alone occupy an intermediate position.

It is evident that as regards the rapidity of disappearance of the infection the combined treatment gives the best results.

If we take into consideration the degree of infection as shown in the columns in the tables it will be found that for the carriers who did not relapse and who were treated with one-grain injections the +++ infections gave an average time of disappearance of 3·5 days, the ++ infections an average of 4·3 days and the + infection 3·5. With the carrier cases treated by the combined method the figures are 2·3, 1·5, and 2·3 respectively.

It thus appears that the intensity of the infection as judged by the appearances in the stool has very little to do with the rate of disappearance of amœbæ or cysts. It is significant, however, that the cases which did not react to the injection of one grain of emetin for twelve days all had large infections, and this is true also to a great extent of the cases which were cured but which relapsed later.

As regards the acute cases, in the majority the infection rapidly disappeared in one or two days, but in a few it persisted for a longer period and in one case as long as the last day of treatment. It is important to note that with all the acute cases the clinical signs of dysentery disappeared very rapidly and this in spite of the fact that practically all relapsed later. Most of these acute cases had already been treated with emetin in other hospitals and had been discharged as cured. The remarkable feature of the emetin treatment is not that in some cases, such as Healy with a +++ infection, there should be a failure to bring about a disappearance of the infection, but that in certain cases, such as Carr, for instance, who had a simply overwhelming infection, the largest we have encountered, the first day of treatment was sufficient to abolish it. It has already been mentioned that the cysts which Carr was

passing produced a fatal dysentery in two kittens. It must be remembered that Healy had a long history of previous dysentery while Carr had none, and it is probably in this direction that an explanation is to be sought. Presumably the degree of involvement of the large intestine was greater and of much longer standing in Healy than in Carr, and in his case it was probably much more difficult for the emetin to gain access to all the amœbæ, many of which would escape in localities such as the necrotic pieces of tissue and the base of old ulcers where there was little or only a poor circulation.

(*k*) *When do Relapses occur?*—In our series of observations we have taken as a period of control one month after treatment has been completed. The majority of our cases have been controlled beyond this period, during which the stools have been examined as a rule on every alternate day. In this way we have been able to detect a relapse as soon as it has occurred. Where relapse has occurred after treatment this has taken place in under twenty days, except in three cases, two of which were carriers treated by injections of one grain of emetin, and one an acute case. In every instance the relapse was judged, as was the diagnosis in the first place, by the appearance of cysts of *E. histolytica* or amœbæ with included red blood corpuscles. It seems evident, therefore, that a control of one month after treatment is quite sufficient to ensure that a cure has taken place. Many of our cases have been subjected to an even longer control.

As regards the acute cases it has been pointed out above that the diagnosis of these was made on the occurrence of amœbæ with included red blood corpuscles in a dysenteric stool and that the treatment in all cases caused the rapid disappearance of symptoms, though the infection might not disappear till later. When these cases relapsed, and this has unfortunately happened in the majority of acute cases treated by us, there is not at first a return of the dysentery, but the relapse has been detected by the appearance of cysts of *E. histolytica* in the stool. One such case (Russell) has been mentioned already. The first sign of relapse was the appearance of cysts in the stool and some time after he was readmitted with acute amœbic dysentery again. In the case of Smith* the relapses could not be judged by the appearance of cysts, for this case never passed any, but by the reappearance of amœbæ with included red blood corpuscles.

(*l*) *The Effect of Previous Dysentery on the Treatment.*—We

* *See note on case, page* 187.

have gone very carefully into the histories of the cases and have obtained information as to previous dysentery. It will be seen by the tables that the majority of carriers gave no history of previous dysentery. Most of them had been on the Peninsula and had had diarrhœa, and those who had had dysentery were, of course, unable to state the kind of dysentery from which they had suffered. Still, the history, whatever it is worth, is set out in Tables X, XI, and XII. Of the thirty-seven carriers treated by one-grain emetin injections and who did not relapse, fifteen gave some history of dysentery, mostly on the Peninsula. Of the ten cases which relapsed, only one had a history of dysentery, while of five who did not react to the treatment two gave a positive history. It seems, therefore, that as far as these carriers are concerned a previous history does not affect the treatment.

When we come to consider the acute cases we find that there was a previous history in every case except three (Rushforth, Wilkinson, and Gaskin), who were suffering from their first attack. These three cases relapsed after a first course of emetin, though a second course was successful in curing one (Rushforth). All the other acute cases gave definite histories of dysentery, generally of repeated attacks. Each had been repeatedly treated in various hospitals with or without emetin. From each place the case had been discharged as cured, but relapse had occurred later on. The histories of these cases are most unfortunate, as the men rarely come back to the same hospital, so that in each instance the same line of emetin treatment is repeated, even if the patient has been fortunate enough to have his condition correctly diagnosed. The danger of emetin in these acute cases is that it produces a false security, for it very quickly abolishes the symptoms without ridding the patient of an infection which can only be detected by expert microscopical examination.

As we have already explained, it seems that the carrier case who has either had no dysentery at all or only a single attack, has a condition of the intestine much more amenable to treatment than that of the case which has repeated attacks of dysentery. The patient whom one sees in the acute dysenteric condition does not differ from the healthy or comparatively healthy carrier, except that he has a more extensive or active ulceration of the large intestine, and if this view is correct it is not surprising that these acute cases are much more difficult to cure than the carriers. In this respect, therefore, history of previous dysentery (if this has been definitely amœbic) or, more especially, histories of repeated attacks as

indicating a more extensive involvement of the large intestine, indicate a bad prognosis as regards emetin treatment.

Exceptions to this rule occur, however, for case Spiers, so frequently referred to in this paper, had a very bad history of recurrent dysentery, yet he was completely cured by emetin.

(*m*) *Effect of Previous Emetin Treatment.*—The possibility of previous emetin treatment having produced a resistant or emetin-fast strain of *E. histolytica* has been discussed above. It will be seen from the table of thirty-seven cases treated by one-grain injections (Table X) that six of the carriers who were cured had had previous emetin treatment for dysentery. Of ten that relapsed, only one had had a previous course of emetin. Of five cases which did not react, two had had previous emetin treatment on many occasions, but these (Healy and Spiers), though in a carrier condition when first seen by us, had just recovered from acute attacks and ought rather to be classed with the actual acute cases. Practically all the acute cases had had repeated attacks of dysentery, and this has meant repeated though less numerous courses of emetin. The three acute cases (Rushforth, Wilkinson, and Gaskin) who had had no previous dysentery and consequently no previous emetin treatment, all relapsed after the first course of emetin; Rushforth and Gaskin were given a second course and the former of these was cured. It would appear, therefore, that a previous course of emetin in itself does not affect the subsequent treatment. The symptoms of dysentery cleared up just as quickly in those previously treated as in those who had had no treatment before.

(*n*) *Delayed Action of Emetin.*—When emetin is administered subcutaneously, on its rate of absorption depends its action on the amœbæ. When a course of injections is given, if absorption does not take place very quickly it is evident that the drug will go on acting on the amœbæ for some time after the treatment has been stopped. Thus with case Greenwood amœbæ were still present in large numbers the day after the course of treatment was completed, but they had completely disappeared at the next examination and they remained absent till a relapse occurred some time later. We do not yet know how quickly the emetin is excreted from the body, but a delayed cure like the one just quoted seems to indicate that the emetin may not reach the amœbæ in its maximum concentration till a day or more after injection.

(*o*) *Best Method of Emetin Administration.*—From what has already been said regarding the cases we have treated it is clear that the best results have been obtained by the employment

of the combined method of emetin treatment. This has consisted in the administration of one grain of emetin hydrochloride subcutaneously each morning, and $\frac{1}{2}$ grain in keratin-coated tabloid orally each night, the course of emetin extending over twelve days. During the course the patient should stay in bed and be kept on light diet, which must be a strict dysentery diet if blood and mucus are present in the stool. It is not necessary to give salines regularly, but they must be employed if there is the slightest tendency towards constipation. This treatment has given us the best results. All the carrier cases treated in this way have been cured, and two, possibly three, out of eight acute cases with definite dysentery. This treatment can, therefore, be safely employed for all cases of *E. histolytica* infection, though the results with acute cases, especially those who have had long histories of repeated dysenteric attacks, will be far from satisfactory when compared with the results obtained in the case of a carrier. The treatment has the effect of almost immediately clearing up the dysentery, while the amœbæ disappear from the stool. In most of the acute dysentery cases, however, relapse occurs later. For this reason we feel that it would be well to treat cases of this nature during long periods as one would treat chronic malaria infections. The initial course of emetin as explained above can be given, and this might be followed by a long course of emetin, say $\frac{1}{10}$ grain, or even $\frac{1}{6}$ grain, taken orally in keratin-coated tabloid each night. A tolerance, as regards nausea, would be quickly acquired, and it is very probable that the daily exhibition of the small dose during two or three months after the initial course of emetin would prevent relapse occurring. We have had no opportunity of testing this method, but from what we know of malaria and quinine there seems to be every prospect of success. Such a small dose as $\frac{1}{10}$ or $\frac{1}{8}$ grain, or even $\frac{1}{4}$ grain, is hardly likely to affect the patient's health, for we know of several cases who have taken much larger doses over long periods without any untoward symptoms developing. We would advise that carrier cases of *E. histolytica* be treated with $1\frac{1}{2}$ grains of emetin as explained above, and that cases with actual dysentery with blood and mucus be treated in the same manner, though with a stricter dysentery diet. If these dysenteric cases show any tendency to relapse, as they frequently do, the emetin course can be repeated and be followed by a long course of emetin by the mouth in small doses as is done with quinine in cases of chronic malaria.

(2) *Treatment of Entamœba coli Infections.*

During the course of the treatment of *E. histolytica* infections by means of emetin a good deal of information regarding the action of this drug on *E. coli* has been obtained. Similarly during attempts to get rid of flagellate infections by means of other drugs the action of these on coincident *E. coli* infections has been observed.

Emetin.—The general difference in the action of this drug in *E. coli* and *E. histolytica* infections has been discussed, and it has been suggested that *E. coli* is probably generally distributed through the large intestine and not so easily acted upon, while *E. histolytica* is more intimately associated with the ulcers and so comes more directly under the action of emetin.

In the series of cases treated by injections of emetin, one grain a day for twelve days, it will be seen that in only one instance (Osgood) did an *E. coli* infection, which was present before the end of the emetin course, fail to appear after the course was completed. It is thus evident that such a line of treatment cannot entirely get rid of an *E. coli* infection. If one looks at the charts of treatment of the individual cases it will be seen that in a few the *E. coli* infection disappears temporarily, apparently as a result of the treatment, but that in others there is no such action whatever. It seems that this line of treatment may influence the infection to some extent, but only very slightly compared with the action on *E. histolytica*.

The administration of a grain of emetin a day by the mouth was no better as regards a permanent result, though the temporary disappearance of the infection was more marked than when the emetin was given by injection.

When we consider the combined injection and oral treatment of $1\frac{1}{2}$ grains of emetin a day, we find there is a much more decided action in the *E. coli* infections and indeed on all other coincident protozoal infections. In nearly every case this line of treatment has abolished all the intestinal protozoa including the *E. coli*. In four cases (English, Liddle, Graham and Smith) there was no return of *E. coli* infection during the period of control. It is evident that, just as emetin by the mouth ($\frac{1}{2}$ grain) combined with injections (one grain) gives better results with *E. histolytica* so it does with *E. coli* infection, but the outstanding feature of the treatment is the specific nature of the action of emetin on *E. histolytica* and its comparative inaction on *E. coli* as regards permanency of cure.

Other Drugs.—Various attempts were made to get rid of flagellate infections, and as these were often associated with *E. coli* it was possible to watch the action of any line of treatment on the amœbæ also. Several cases were treated with turpentine (ten minims three times a day) in the form of mist. terebinth. There was no action on *E. coli.* Similarly *E. coli* proved refractory to bismuth salicylate twenty grains three times a day alone, and in combination with beta-naphthol fifteen grains three times a day. The latter drug alone likewise had no effect on the *E. coli* infections. A course of saline purging will do much to get rid of an *E. coli* infection and a subsequent examination of the stool may give negative results for some days. The absence, however, is only apparent, for the infection invariably reappears later on. Similarly attacks of diarrhœa or dysentery with much purging will mechanically wash away the majority of the amœbæ, so that an infection may apparently disappear. This is shown very clearly by the number of such cases which we found to be negative at first and which became positive as the stools began to approach normal. On the other hand, certain diarrhœic conditions favour the multiplication of *E. coli* as they do other intestinal protozoa, sometimes with the result that a large infection may persist throughout such an attack. In one such case already mentioned the diagnosis was in doubt for some days, for numerous amœbæ were passed constantly in a diarrhœic stool and it was only after the lapse of about ten days that the stools returned to normal and cysts of *E. coli* became numerous.

(3) *Treatment of Lamblia Infections.*

We have made numerous attempts to rid cases of their lamblia infections, but though many of the drugs tried will abolish an infection as judged by stool examination the infection almost invariably reappears.

Emetin.—This drug given in the form of injections of one grain a day for twelve days will sometimes cause a lamblia infection to disappear for a time, but given by the mouth emetin will nearly always produce a temporary clearing up of the infection. Cases Gildel, Amers and Stone, whose charts of infection are given at the end of this paper, illustrate this general rule very well. In practically all these cases the infection returns later. In one case (White), however, the infection disappeared and there was no return during a control of forty days. This case, however, was given a course of beta-naphthol fifteen grains with bismuth salicylate twenty

grains three times a day for twelve days after the course of emetin was finished.

Beta-naphthol in a dose of fifteen grains three times a day was tried in several cases without any good result. Case Gildel was one of these. The drug did not even cause the parasites to disappear.

B. naphthol and Bismuth Salicylate.—These two drugs in the form of a powder in the proportions just mentioned above have been given to a number of cases of lamblia infection. In all these the infection has disappeared after a varying number of days, sometimes after two days' treatment, at other times after seven or eight days. So constant has been the disappearance of lamblia cysts from the stools of cases that it is difficult to believe that the drug has no action on the infection and that the vanishing of the cysts is the result of the natural course of the infection. Unfortunately, however, the cure is only a temporary one, as the infection almost invariably reappears after a week or two of control. Only in the single case mentioned on *page* 128 under the emetin treatment did it appear that a cure has been effected, but whether this was the result of the emetin or the subsequent treatment cannot be stated.

Bismuth Salicylate.—This drug alone in a dose of twenty grains three times a day was used on several cases. It was found that the action was just as certain as when the beta-naphthol was used with it. Apparently the action of the former mixture was due to the bismuth salicylate rather than to the beta-naphthol. Bismuth salicylate will usually cause a lamblia infection to disappear and thereafter the diarrhœa with associated mucus will stop also. One must not forget, however, that the natural course of a lamblia infection is an intermittent one and that the symptoms described above are intermittent also and will subside without any treatment whatever. It is this fact which makes a judgment on the action of a drug so difficult.

Case Baker is very interesting in this respect. He was a carrier of *E. histolytica* who was treated by the combined oral and injection administration of emetin. Three days after the course of emetin was completed a lamblia infection, not hitherto evident, made its appearance. The case was controlled for a month and as the *E. histolytica* infection did not reappear the man was discharged though lamblia cysts were still present in the stool. Five weeks later he was readmitted to hospital for colitis. The stool was examined on several occasions but there were present no lamblia cysts. Whether this was a case of spontaneous cure or whether the case was passing through one of the periods when the lamblia

infection (as judged by the cysts in the stool) was in abeyance cannot be stated. The case serves to illustrate the difficulties associated with the control of cases of this kind where the infections only reveal themselves intermittently in the stool.

It appears, therefore, that no very satisfactory treatment for lamblia infection exists. For general purposes the treatment by bismuth salicylate seems to be the best and this drug can be given for long periods without any harmful effects. It has the advantage of clearing up the symptoms associated with lamblia infection even if it does not permanently get rid of the parasite.

It might be thought that a flagellate living in the small intestine could be attacked much more readily than one in the large intestine. As a matter of fact drugs seem to act more readily on the lamblia infection, but as explained above the relapse almost always occurs. An examination of animals infected with lamblia by sectioning the gut, has shown that the flagellates live not only on the surface of the intestine but that many of them make their way into the glands, where they can be seen in rows on the glandular epithelium. It is possible that they may spread into the bile ducts, but we have no information on this subject. The important point in treatment and the one which makes it so difficult is that intestinal disinfectants act directly on the flagellates in the gut lumen but not on those within the glands. These escape destruction and eventually when treatment is stopped re-establish the gut infection. This hypothesis affords an explanation of the action of drugs like emetin and bismuth salicylate, which cause a temporary disappearance of infection as judged by stool examinations.

(4) *Treatment of Tetramitus Infection.*

The action of emetin on this flagellate has been observed in several cases of *E. histolytica* infection. While it can be stated that emetin in the form of injections of one grain a day for twelve days has no action on the flagellate, emetin administered orally has a decided action. In the case of Gildel who passed tetramitus regularly for three months, the administration of $\frac{1}{2}$ grain of emetin by the mouth for twelve days caused it to disappear. It returned, however, some days after the course was completed. In practically all the cases of *E. histolytica* infection when tetramitus was present also, the latter disappeared during the course of treatment by the combined method of injection and oral administration ($1\frac{1}{2}$ grains a day). In only one case did it fail to reappear during the subsequent control of the case.

Beta-naphthol.—This drug was used in the case of Gildel mentioned above; fifteen grains were given three times a day for twelve days without any change in the tetramitus infection.

Turpentine.—Turpentine in the form of mist. terebinth. with a dose of ten minims of turpentine three times a day was tried. In one case, Peacock, the infection disappeared during the treatment, but it reappeared eight days after the course. In another case, Fulford, it had a similar effect, while in another, Buckley, the flagellates did not even disappear from the stool. It seems doubtful if turpentine given in this way can have any permanent effect on a tetramitus infection.

Bismuth Salicylate.—In a dose of twenty grains three times a day bismuth salicylate was tried on several cases. In some of these during the course of treatment the tetramitus disappeared while in others there was no such disappearance. Owing to the irregular course of these infections it is difficult to pronounce any definite opinion on the action of this drug, for we have not controlled these cases sufficiently. A subsequent control of less than one month after treatment is insufficient to enable any result to be claimed as a permanent cure.

It is evident that though certain drugs will cause a tetramitus infection to disappear, there is nearly always a subsequent relapse. No satisfactory treatment for the infection is known.

(5) *Treatment of Trichomonas Infections.*

Naturally enough, with an infection which runs such an intermittent course, it is exceedingly difficult to judge the action of any line of treatment. From the tables of the cases treated by emetin where trichomonas infections co-existed it does not appear that this drug has any action on the flagellate.

Turpentine.—This drug in a dose of ten minims three times a day (in mist. terebinth.) has been tried in several cases of trichomonas infection. One of these cases (Ruane) had an infection in a soft unformed stool. The mixture was taken for twelve days and trichomonas was last seen the day before the end of the course. The stools were examined on eight occasions during the next month, but no flagellates were found. It must be stated, however, that during the period of control the stools were formed, so that the flagellate may still have been present in the gut though not in the stool. In another case (Fulford) where there was a mixed infection of trichomonas, tetramitus, lamblia and *E. coli*, the first named flagellate disappeared during the course of mist.

terebinth. but reappeared afterwards. It is thus manifestly impossible to tell if the turpentine was or was not responsible for the disappearance of trichomonas in the first case or for its temporary disappearance in the second.

Bismuth Salicylate.—This drug has been given in doses of twenty grains three times a day to cases of trichomonas infection, but the results we have obtained are as indefinite as with the other treatment employed.

(6) *Treatment of Coccidial Infections.*

As we have stated above, only one case of this infection (Isospora) was encountered by us in Egypt. This was in a carrier case of *E. histolytica*, and as we have already explained, one grain of emetin subcutaneously administered for twelve days failed to effect a cure of either the *E. histolytica* or coccidial infection, whereas a second course of emetin, one-grain injection and ½ grain by the mouth, caused both infections to disappear and no relapse occurred. If we may judge from this single case, it may be assumed that emetin had some action in getting rid of the coccidial infection.

(7) *Treatment of I-cyst Infections.*

Several of the cases of *E. histolytica* infection have had I-cysts present also, so that we have been able to note the action of emetin on these. Both emetin injections (one grain a day) and emetin by the mouth appear to cause the I-cysts to disappear from the stool. We have not followed any single untreated I-cyst infection for long, so we cannot be quite sure that the disappearance was not accidental, but under the emetin treatment the I-cysts vanish after the second or third day of the course and they do not recur during the subsequent control of the case. It seems reasonable to assume that the emetin was the cause of their vanishing. As, however, we know nothing of the behaviour of I-cysts in the intestine and as they do not appear to be pathogenic in any way unless their occasional presence in large numbers in the fæces of sick people is a sufficient argument in favour of pathogenicity—the question of the treatment is not one of practical importance.

(8) *Treatment of Blastocystis Infections.*

We have no definite information to offer as regards the action of drugs on blastocystis. Emetin by the mouth combined with injections has the power of clearing away all protozoal infections of the gut, and with these vanish, as a rule, the blastocystis infection as well.

Summary of Matter Discussed in Part III.

(1) The cases treated by emetin hydrochloride fall into three groups, all of which were treated for twelve days : (1) those treated with a daily one-grain injection of emetin ; (2) those treated with a daily one-grain dose of emetin by the mouth ; and (3) those treated with a daily one-grain injection of emetin together with half a grain dose by the mouth. The result is based on a control of each case for at least one month after treatment.

Under group 1 were treated fifty-two carriers. Of these 37 were cured, 10 relapsed, and 5 showed no reaction to treatment. Six acute cases all relapsed after treatment.

Under group 2 were treated nine cases. Of these 6 were cured, 2 relapsed, and 1 showed no reaction to treatment. Of three acute cases, two relapsed and one did not react to treatment. One very chronic case was cured by emetin $5\frac{1}{2}$ grains by the mouth during seven days.

Under group 3 were treated thirty carrier cases. Of these all were cured. Of seven acute cases two were cured and five relapsed.

(2) Methyl emetin sulphate in a dose of two grains a day for twelve days (one-grain injection each morning and one grain by the mouth each night) failed to cure two acute cases but was successful in curing one carrier case. Methyl emetin sulphate does not produce vomiting so easily as emetin hydrochloride.

(3) One case which did not react to emetin hydrochloride was treated without result with thymol and pulvum ipecacuanha.

(4) In the treatment of carriers the object is to kill the amœbæ and not the cysts which are harmless to the individual who passes them.

(5) A comparison of the three lines of treatment adopted shows that the best results are obtained by the combined injection and oral administration of emetin (a total of $1\frac{1}{2}$ grains of emetin daily).

(6) It is evident that the healthy or comparatively healthy carrier is much more easily cured of his infection than the acute case with actual dysentery, especially when there is a history of repeated attacks of dysentery. It is possible that in some long standing cases the condition of the ulcers in the intestine prevents the emetin reaching the amœbæ.

(7) It appears at first sight that in certain cases emetin-resistant strains of amœbæ exist. The resistance is, however, only apparent, and is probably due to the emetin not having reached the amœbæ.

(8) There is no evidence to prove that injections of emetin tend to make the amœbæ encyst or to increase the number of carriers.

(9) The action of emetin on *E. histolytica* is not influenced by any coincident protozoal infections.

(10) In the case of carriers it is doubtful if rest in bed and diet assist the emetin in its action on *E. histolytica*. Rest in bed, however, is useful in preventing ill-effects of emetin on the patient. Acute cases should be kept in bed on dysentery diet.

(11) It is doubtful if salines regularly administered influence the emetin treatment in any way.

(12) Emetin may produce irregular action of the heart when given in large doses. Administered by the mouth emetin readily causes vomiting, which, however, does not hinder the action of the drug on the amœbæ.

(13) All the treated cases which have relapsed have done so in under twenty days after the completion of the course, with the exception of three which relapsed in under thirty days.

(14) A previous history of a single attack of dysentery (which may or may not have been bacillary but most probably the latter) has very little effect on the treatment of carrier cases. Cases which have suffered from repeated attacks of dysentery are difficult to cure. A history of repeated attacks would indicate an amœbic rather than a bacillary infection.

(15) Previous emetin treatment does not affect the subsequent treatment unless it means that there has been a long history of repeated attacks of dysentery.

(16) Emetin injected under the skin may be some time in reaching the amœbæ, so that its action may be delayed.

(17) As acute cases of amœbic dysentery so frequently relapse after emetin treatment it is suggested that after the course of emetin ($1\frac{1}{2}$ grains subcutaneously and orally administered) the case might possibly be given a long course of emetin in small doses by the mouth as is done in the case of malaria and quinine or trypanosomiasis.

(18) Emetin by the mouth will frequently get rid of *E. coli* infections but relapse almost invariably occurs.

(19) Emetin by the mouth will also temporarily get rid of flagellate infections (lamblia, tetramitus and trichomonas). Bismuth salicylate will also abolish these infections, but relapse occurs after short courses. Longer courses might give good results.

(20) Both emetin injections and emetin by the mouth appear to cure I-cyst infections. Emetin by the mouth appears to have some action in abolishing a blastocystis infection. One case of coccidium (isopora) infection appeared to be cured by emetin $1\frac{1}{2}$ grains a day administered orally and by injection.

PART IV.*

Experimental Work with the Human Intestinal Protozoa, their Carriage by House-flies and the Resistance of their Cysts to Disinfectant and other Agents.

(1) *Experimental Work.*

A number of experiments were conducted with cats, rats and mice with a view to infecting them with intestinal protozoa. Most of these gave negative results but are none the less interesting. The following is a record of these.

(1) *E. histolytica.*—An attempt was made to infect two kittens by means of flies which had fed on fæces containing *E. histolytica* cysts. Batches of flies were allowed to feed on fæces and then placed over bread and milk on which they deposited fæces very plentifully. The kittens were then fed on the bread and milk. This was repeated daily for six days, after which the kittens were carefully observed. Between three and four weeks later the kittens became ill and a couple of days after passed typical dysenteric stools with blood and mucus. Examination of this microscopically showed numerous cells of all kinds but no amœbæ. The kittens recovered and one was killed ten days later. There were no lesions of the gut and no amœbæ were found. The nature of the dysenteric attack was thus undetermined.

(2) Two cats were anæsthetized and inoculated intrahepatically with about two to three cubic centimetres of liver abscess pus containing numerous active *E. histolytica*. One cat escaped but continued for some weeks to wander about the hospital grounds. It remained perfectly well and active enough to avoid capture. The other cat remained well for some time, but twenty-six days after the inoculation it became ill and lethargic. The next day as it was worse it was chloroformed. No lesions of any kind were discovered and there was no mark of the inoculation, though there was no doubt whatever that this had actually been made into the liver.

(3) A kitten was inoculated *per rectum* with about ten cubic centimetres of liver abscess pus containing active *E. histolytica*.

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* Reprinted from the *Journal of the Royal Army Medical Corps*, June, 1917.

As nothing happened, a week later the injection was repeated. No dysentery developed and no infection occurred.

(4) A young kitten was given about five cubic centimetres of fæces emulsion of case Carr with large infection of *E. histolytica* cysts. Case Carr had never suffered from dysentery. The emulsion was administered by pouring it into the kitten's mouth. The kitten with the mouth still soiled was placed in a cage with another kitten. A week later both kittens became ill with acute dysentery, from which they died. There were numerous *E. histolytica* in the blood and mucus stools and extensive ulceration of the large intestines. The experiment is of interest in showing that cysts from a carrier who had no dysentery could produce acute dysentery in cats. The second kitten must have been infected by licking cysts from the soiled mouth of the first cat.

(5) A kitten was given *per os* on two occasions emulsion of fæces of case Healy. There was a history of repeated attacks of dysentery and numerous cysts and minute forms of *E. histolytica* were present. The kitten did not become infected. In this instance, though there was a definite dysentery history, no infection of the kitten took place (compare Experiment 4).

(6) A kitten was given on two occasions *per os* emulsion of fæces of case Smith, who constantly suffered from amœbic dysentery, there being present in the stool active amœbæ, many of which contained red blood corpuscles. Cysts of *E. histolytica* were never found in this case during a long observation (see above). The kitten did not develop dysentery and did not become infected.

(7) Two white rats were fed on two successive days with fæces emulsion containing numerous *E. histolytica* cysts. The rats did not become infected and showed no signs of illness during an observation of over two months.

(8) A mouse was fed on several occasions with fæces of case Healy containing numerous *E. histolytica* cysts. No infection and no sign of illness was noted in an observation of over three months

Tetramitus Mesnili.—(1) A large quantity of emulsion of fæces containing numerous free and encysted tetramitus was introduced into the stomach of a kitten by means of a stomach tube. The kitten never showed any tetramitus infection.

(2) A mouse was fed on the same material but no infection occurred.

(3) A rat was similarly treated and likewise did not become infected.

Coccidia (*Isospora*).—(1) A kitten was fed on several occasions

with developed oöcysts of the human isospora. No infection took place. An isospora is found very commonly in Alexandria cats, but the oöcyst is quite unlike that of the human parasite. The oöcysts of the cat isospora resemble those of the European form.

(2) A mouse was fed repeatedly with developed oöcysts and no infection took place during an observation of over four months.

Observations on Lizards.—A number of lizards which lived in open spaces in Alexandria were examined. Two distinct types were dissected. One of these (Agama sp.?) is very common in stony places, where it lives on flies and also appears to feed upon vegetable material. In this amongst other protozoa were found a tetramitus and an amœba which resembled *E. coli* not only in its free stage but also in the production of an eight-nuclear cyst indistinguishable from that of *E. coli*. The other lizard was a skink which lived in sandy places. It fed exclusively on insects. There were numerous flagellates in the gut but none resembling those of the human intestine. It seems just possible that lizards might become infected with human parasites by feeding on flies which had already fed on human fæces.

(2) *Flies as Carriers of Intestinal Protozoa and other Infections.*

The relation of house-flies to the human intestinal protozoa and the possibility of their dissemination by flies which have fed on infected fæces has been discussed by us in two earlier publications. (Memorandum on the carriage of cysts of *E. histolytica* by house-flies, with some notes on their resistance to disinfectants and other agents, issued by the Medical Advisory Committee, Mediterranean Area, April, 1916, and the same with additional notes on a more extended examination of wild flies, in the *Journal of the Royal Army Medical Corps*, May, 1917.)

We have shown that flies which feed on fæces containing the free or encysted protozoa readily take these into their intestine. By dissecting flies at various intervals after feeding we have noted that so long as any of the fæces remained in the gut the encysted forms could be found.

The following is a record of some of our observations. In these nothing of a doubtful nature has been accepted as evidence of the presence of a cyst of one of the intestinal protozoa. We have carefully ignored anything which was not absolutely certain, so that our finds in the examination of wild flies are somewhat lower than was actually the case.

(*a*) *Flies fed on Infected Fæces and dissected later.*—(1) Six

flies were enclosed under a glass with fæces of case Ure which contained a fair number of cysts of *E. histolytica* and a leptothrix. The flies were dissected twenty hours after the fæces had been removed. The stomachs of all the flies contained fæces in which cysts of *E. histolytica* occurred as well as the leptothrix which was present in the stool. The loaded rectum of one fly was cut off and examined separately. A few cysts of *E. histolytica* were seen. Some of the cysts appeared quite normal and did not stain with eosin, others appeared to be degenerate. In our previous publications we have shown that cysts which do not stain with eosin are probably alive.

(2) The same experiment was repeated with fæces from the same case two days later with three flies. Dissected eighteen hours after feeding, all three flies showed fæces in the gut, in which the leptothrix was present but nothing definitely diagnosable as cysts of *E. histolytica* were found.

(3) Twelve flies were allowed to feed on fæces of case Turner containing a fair number of cysts of *E. histolytica*. Dissected eighteen hours after feeding the flies gave the following results: (*a*) One fly had a fairly full gut and in the contents four undoubted cysts were found. (*b*) Six flies had practically no fæces in the gut and no cysts were discovered. (*c*) Five flies had absolutely empty guts and no cysts were found.

(4) Eight flies were fed on fæces of case McCaffrey which contained a large number of cysts of *E. coli* and a small number of cysts of *E. histolytica*. The flies were dissected twenty-four hours after feeding with the following results: (*a*) Four flies had empty guts and no cysts were found. (*b*) Two flies had a little fæces in the gut and eight-nuclear cysts of *E. coli* were found. (*c*) One fly had a good quantity of fæces in the gut, a fair number of cysts of *E. coli* were present and one cyst of *E. histolytica* was seen. The cyst did not stain with eosin. (*d*) One fly had a good quantity of fæces in the gut and numerous cysts of *E. coli* which did not stain with eosin.

(5) Three flies were fed on stool of case McCaffrey and were dissected forty-two hours after feeding. (*a*) One fly had a fair amount of fæces in the gut and a corresponding number of *E. coli* cysts which did not stain with eosin. (*b*) Two flies had empty guts and no cysts were found.

(6) Three flies were fed on stool of case McCaffrey and were dissected seventy hours after feeding. The gut was empty in all three flies and no cysts were discovered.

(7) Nine flies were fed on fæces of case Hancock with a large number of cysts of *E. histolytica* present. The flies were dissected forty-two hours after feeding. In every case the gut was empty and no cysts were discovered.

(8) Eight flies were fed on fæces of case Badham which contained large numbers of cysts of *Lamblia intestinalis*. The flies were dissected twenty-four hours after feeding with the following results: (*a*) One fly had no fæces in the gut and no cysts were found. (*b*) Two flies had very little fæces and no cysts were found. (*c*) Five flies had a good amount of fæces in gut and numerous lamblia cysts were present. With eosin some stained and others did not.

From the foregoing records of feeding experiments it is clear that flies readily take up cysts when they feed on fæces, and that these cysts remain in the gut so long as fæces remain there. The flies, however, get rid of the fæces in twenty-four hours, after which cysts can no longer be found. In the experiments recorded above the flies were given no food after having fed on the fæces. It is probable that the fæces would be more quickly got rid of if the flies were feeding constantly as they do in nature. Further, the cysts do not degenerate to any extent; at any rate, the length of time the cysts would remain in the gut under natural conditions would not be enough to bring about their degeneration.

(*b*) *Flies fed on Infected Fæces and their Fæcal Droppings examined later.*—(1) One fly was allowed to feed on fæces of case Hancock with large infection of *E. histolytica* cysts. Half an hour later six dried fæcal deposits were taken up in eosin saline solution. There were present a fair number of cysts of *E. histolytica*, all of which were stained. A further number of droppings were taken up two hours later with a similar result.

(2) Four flies were allowed to feed as above. Two hours after the fæces had been removed the flies were given fresh fæces on which to feed. Soon after feeding some moist fæcal droppings were taken up in eosin saline. There were present fair numbers of cysts of *E. histolytica* which did not stain with eosin.

(3) Six flies were fed on fæces of case Hancock with large infection of cysts of *E. histolytica*. Sixteen hours after the removal of the fæces the flies were given sugar and water on which to feed. The droppings which were deposited soon after were taken up while still moist in eosin saline solution. There were present fair numbers of cysts of *E. histolytica* which did not stain with eosin.

(4) Six flies were fed on fæces of case Hancock with large

infection of cysts of *E. histolytica*. Moist droppings of the flies were examined twenty and thirty minutes later, and cysts of *E. histolytica* were easily found in both examinations.

(5) Six flies were allowed to feed on fæces containing a mixed infection of *E. coli* and lamblia cysts. Moist droppings deposited forty minutes later contained cysts of *E. coli* and lamblia.

(6) Experiments of the same nature were conducted with *Calliphora* sp. and *Lucilia* sp., with similar results.

(7) Six house-flies were allowed to feed on liquid fæces containing active trichomonas. Moist droplets were passed within five minutes of feeding, and these contained living unaltered trichomonas.

(8) A small batch of flies were allowed to feed on the fæces of a case containing cysts of lamblia (numerous), and *E. histolytica* (few) ; after five hours the dried droppings of these flies were examined. Numerous lamblia cysts were found, and in addition a cestode egg (40 by 48 microns), and an operculated trematode egg (20 microns). These flies had been caught near the laboratory where a cat was kept, which was known to be passing similar cestode and trematode eggs in the fæces.

The above experiments were conducted with ordinary wild flies. They were placed in glass globes covered with mosquito netting, and were allowed to starve for a few hours before the fæces were offered to them. This was done by placing a small quantity on a cover-glass and sliding it under the jar. When the flies had fed it was removed. To obtain the droppings before they dry it is necessary to watch the flies carefully as the droppings dry very quickly, especially in a hot country where the observations were made. A long drawn-out capillary pipette is used and some saline is run into the fine capillary end of this. So soon as a fly is seen to deposit a dropping a tiny drop of saline is blown on to it and it is then touched with the capillary end of the pipette, when it will run up into the capillary tube. As much of the contents of the capillary tube as is necessary is then blown on to a slide and examined under a cover-glass. If there is any doubt iodine solution can be added.

This method of examination of moist droppings is very useful for the control of the passage of infectious material through flies, and it could very readily be adapted for bacterial work in connection with the passage of typhoid, dysentery and other bacteria through the flies' intestine.

(*c*) *Examination of Wild Flies taken in various Localities.*— The main result of our examination of wild flies, as published in

the *Journal of the Royal Army Medical Corps*, May, 1917, is reprinted on *pages* 149-155, but some further explanation of the methods employed will be given here together with some additional observations.

The majority of the flies were examined singly, though some were examined in batches. When examined singly, the fly was caught in a glass tube (2 by 1 inches), and the tube was placed on the laboratory bench standing with the open end on a glass slide. No food was given to the fly, which deposited droppings either on the sides of the tube or on the slide. The total number of the droppings deposited by the fly, the number of droppings on the slide, and the length of time since capture were noted. Only the droppings on the slide were examined. In most cases these were dry when examined, but the drying did not interfere seriously with the identification of the objects present. The examination was carried out by running some saline solution on to the slide, and placing over the droppings a cover-glass. The droppings were then examined with the $\frac{2}{3}$ and $\frac{1}{6}$-inch objective without disturbing them. A gentle tap on the cover glass then caused the droppings to spread out slightly, when such objects as worms' eggs could be detected. From one large dropping there emerged in this manner two lateral-spined bilharzia eggs. Finally, the cover-glass was moved, so that the droppings were completely emulsified in the saline, and search was made with the $\frac{1}{6}$ and $\frac{1}{12}$-inch objectives for cysts of protozoa and other objects. It is evident that any eggs or cysts discovered in this manner must have passed through the intestine of the fly. The view that any of the cysts or eggs found had been transported on the legs of the flies is hardly tenable. As far as the worms' eggs are concerned, these were always found in the droppings themselves before they had been completely emulsified in the saline—and this was true most usually of the cysts also—so there is very little likelihood that any of the objects found had been deposited by the flies' legs on the slide between the droppings.

As already stated, doubtful objects were ignored, and, for the identification of the cysts, iodine solution was often used by drawing it under the cover-glass by means of blotting-paper.

We have given our method of examination in some detail, because some other observers who have examined flies have relied upon dissection of the gut, and quite recently Shircore, who has examined flies for worms' eggs in Mombassa Hospital, employed a method which involved emulsifying the organs of the flies, and centrifuging after addition of ether. The examination of droppings

as we have done affords a very simple method for detecting to what extent flies in any locality are infecting themselves through having access to fæces. Further, it is indirectly an indication of the efficiency of the sanitary arrangements in any neighbourhood where human beings are infected with organisms which can be recognized microscopically in the stool.

It is impossible to give a complete record of all the flies examined with the number of droppings passed by them, and the time occupied. This will be done only for the flies which were found to be positive by way of illustrating the records which have been kept. (*See* Table.)

TABLE SHOWING RESULTS OF EXAMINATION OF 18 POSITIVE FLIES OUT OF A TOTAL OF 229.

Number	Total droppings	Droppings examined	Time	Result
1	9	9	8 hrs.	One cestode egg with six-hooked embryo μ40; one trematode operculated egg μ30; (probably *Heterophyes heterophyes*).
3	13	13	8 ,,	E.c.c. (several cysts)
9	20	3	6 ,,	E.c.c. (several cysts), E.h.c. (one cyst)
29	7	2	10 ,,	Two lateral-spined Bilharzia ova μ130 × 50 (in one dropping)
38	4	4	4 ,,	E.c.c. (one cyst), E.h.c. (one cyst) and ovum of *Trichocephalus trichiurus.*
51	6	3	4 ,,	Trematode ovum μ35 × 20.
84	2	2	15 mins.	E.c.c. (one cyst)
109	2	2	4 hrs.	E.h.c. (one cyst)
144	4	4	20 mins.	*Tænia saginata* egg.
135	5	3	2 hrs.	Coccidium oöcyst μ28 × 20 (Eimeria).
142	2	2	10 mins.	Lamblia cysts in great numbers, 36 in one dropping.
150	2	2	1 hour	Possibly nematode egg μ44 × 20.
151	1	1	2 hrs.	E.c.c. (one cyst)
153	2	2	2 ,,	E.h.c. (one cyst)
155	4	4	2 ,,	E.c.c. (several cysts), and three ankylostome ova.
167	3	2	3 ,,	Possibly nematode eggs μ40 × 20.
194	7	5	3 ,,	Egg of *Trichocephalus trichiurus.*
216	7	4	7 ,,	E.h.c. (one cyst).

All the above flies were caught in the hospital compound, and many of them in the neighbourhood of, or actually inside, the cook-house, which was built against a wall separating the hospital compound from a native village. This village was in a filthy condition, and the natives were constantly depositing fæces along the front of this wall. It seems clear that the flies must have become infected in the village on the other side of the wall.

In this manner there were examined in all 229 flies, with the results given in the table on *page* 142.

In addition to the examination of single flies, a number of flies were examined in batches, the collective droppings of each batch being examined without any reference to which fly of the batch had deposited the dropping. Nine batches of 6, 8, 3, 5, 3, 3, 2, 2, 4, flies were examined in this way, with the result that a single ankylostome egg (60 by 40 microns) was found in a dropping of the first batch of flies.

Two calliphora, two sarcophaga, and one lucilia deposited seven droppings in an average of three hours and a half. Nothing was found in the droppings.

As regards the 229 flies which were examined singly, the total number of droppings deposited by these was 1,470, of which 608 were examined. The average time each fly remained in the tube before the droppings were examined was four hours and a half. The average number of droppings of each fly is between six and seven in this interval of time. In another experiment with wild flies twelve were enclosed in a box made of glass slides. During the first twenty-four hours after capture the flies deposited in all 283 droppings, given an average of 23·5 for each fly. The flies were given no food after capture.

(*d*) *Quantity of Fæces taken up by Flies.*—An attempt was made to obtain some indication of the quantity of fæces taken up by a fly in a limited period of time. To this end a series of weighing experiments were carried out in the following manner. Small quantities of fæces were placed in cover-glasses, and these were weighed. Two cover-glasses of approximately the same weight were used in each observation. They were weighed the one immediately after the other, and were then placed under two glass globes, in one of which were one or more flies which had been without food for two or three hours. After half an hour's exposure to the flies, the cover-glass was again weighed, and the loss in weight noted. The control cover-glass in the second globe without flies was then weighed, and the loss in weight by evaporation deducted from the loss in weight of the first cover-glass. It was assumed that as the weight of cover-glass and fæces was approximately the same in the two cases, the loss by evaporation would be the same, or nearly so.

As a result of seven experiments in which sixty-one house-flies were used the following figures representing weight in grammes were obtained for the quantity of fæces taken up by a single fly :

0·001, 0·0005, 0·0003, 0·001, 0·001, 0·0027, 0·0024. This gives an average of 0·001 gramme per fly. It may be assumed, therefore, that a single fly which has not fed for two or three hours can take up one milligram of fæces in half an hour.

General Considerations.

In our former communications we have emphasized the influence of the house-fly as a disseminator of infectious material. The experiments show how readily this is done, for a fly which is constantly feeding is constantly passing material through its gut, and this may be accomplished in five minutes as proved by the trichomonas experiment recorded above where the living flagellate was found in the droppings of the fly five minutes after feeding. It is quite evident that as the unprotected flagellate can pass safely through the intestine of the fly encysted stages of protozoa will do so much more easily. In fact, one can safely assume that all such organisms, including bacteria, will in such a short time pass undamaged through the fly's intestine. This being the case, it seems that the fly is much more dangerous on account of material passed through its intestine directly than on account of material which it may regurgitate, or which has become adherent to its legs or body, where it quickly dries and is in most cases quickly destroyed. There is no question of any development in the gut of the fly, which acts merely as a distributor of infectious material. In warm countries there is great danger from this, for these flies abound, and there the insanitary native is in league with the flies, for, by depositing his fæces indiscriminately in the open, he not only supplies infectious material for the flies to feed upon, but at the same time affords them a breeding-ground wherein they can lay their eggs.

We have shown above by the record of our examination of natives in the Hadra prison and of a small number of human fæcal deposits collected in the open how common are protozoal infections amongst the natives, and it is not to be wondered at that we have found such a comparatively large number of infected flies, especially when it is remembered that these flies had probably been feeding previously in the native village. In these countries flies must be constantly taking up material and depositing it upon food, and it seems to us that the wide distribution of the intestinal protozoal infections amongst the natives can more readily be accounted for in this way than any other. It is probable also that other intestinal disorders are spread in a similar manner.

The observations recorded demonstrate the importance of all measures directed against the flies, their capture and destruction and the removal of every possible breeding place. It is perhaps of interest to record here the fact that one of the large fly-traps designed by Lieutenant-Colonel Balfour, C.M.G., R.A.M.C., was put up in the hospital compound near the cook-house. It had a marked effect in reducing the number of flies which entered the cook-houses, and this can easily be understood when we realize that a catch of forty-eight hours yielded one and three-quarter pints of flies. A count was made and it was found that one pint of flies (mostly Musca, with an admixture of larger forms such as Calliphora, Lucilia, Sarcophaga, etc.), numbered a little over ten thousand. Furthermore, many of the flies recorded in the table above as being infected, were actually caught either inside or as they were about to enter the fly-trap.

Conclusions regarding the Fly Question.

(1) Flies feeding on fæces readily take up encysted and other forms of protozoa into their intestine.

(2) The encysted forms of the protozoa can be found in the fly's intestine so long as any fæcal matter remains there. If the flies are prevented from feeding this may be as long as forty-two hours. If the flies are feeding constantly off various materials the later feeds tend to clear out what has been taken up before, so that the time becomes shorter.

(3) The cysts do not degenerate to any extent in the gut of the fly.

(4) Flies can deposit in their own droppings material they have ingested only five minutes before. Live trichomonas were found in the fæces of a fly which had only fed five minutes before.

(5) Cysts of protozoa (and eggs of worms) can readily pass unaltered through the intestine of the fly.

(6) Under natural conditions, if flies have access to infected fæcal material (cysts of protozoa or eggs of worms) a large percentage of the flies taken in the neighbourhood, as proved by the examination of their droppings, will be found to have the infectious material in the gut and a still larger proportion will be found to have fed on fæcal matter.

(7) Flies becoming infected in this way will naturally deposit the material on any kind of food on which they feed, and it seems that the wide distribution of human protozoal infections in warm countries can best be explained in this way.

(8) The direct passage of practically unaltered material through the gut of the fly would seem to be of more importance in the distribution of disease generally than the regurgitation of such material through the proboscis or its adherence to the legs or bodies of the flies.

(9) These observations all tend to emphasize the importance of the well-known sanitary measures directed against the fly, its destruction by traps and other means, the removal of its breeding places, the protection of food, kitchens and dining rooms and latrines from its inroads, and the removal of the dwellings of natives as far as possible from those of the Europeans.

(3) *Resistance of Cysts of E. histolytica and other Intestinal Protozoa.*

In our former publications we have described our experiments on the action of various reagents on cysts of *E. histolytica*. By way of making the present report complete, we will enumerate the chief conclusions here. We have explained that the eosin test as applied by Kuenen and Swellengrebel seems to be reliable. Cysts which stain with eosin are dead and those which resist the stain are alive. This was the test we applied in determining the viability of cysts.

(1) Cysts of *E. histolytica* will survive for over a month in water provided there is great dilution of the fæces.

(2) Cysts of *E. histolytica* will not withstand drying but are killed instantaneously. The Thompsons have shown that the free-living *Amœba limax* produces a cyst which will withstand complete and prolonged desiccation under a tropical sun.

(3) Cysts of *E. histolytica* are killed at once if fæces containing them are mixed with an equal quantity of 1 in 10 cresol solution. Free chlorine in water to a strength of 1 in 10,000 has no effect on the cysts even after several hours' exposure.

(4) Cysts of other intestinal protozoa behave in a similar manner.

The inference is that the intestinal protozoa will spread from man to man only if the encysted stages remain moist, and this condition is fulfilled in fly and water carriage.

Summary of Matter discussed in Part IV.

(1) Attempts were made to infect rats, mice and kittens with *E. histolytica*, both in fæces and liver abscess pus. Two kittens alone became infected.

(2) *Tetramitus mesnili* free and encysted failed to infect a rat, a mouse and a kitten.

(3) A kitten and a mouse failed to become infected with the human coccidium (Isospora).

(4) Lizards (Agama sp.?) harbour tetramitus and an amœba. The latter resembles *E. coli*, both in the free condition and the production of an eight-nuclear cyst.

(5) House-flies readily take up free and encysted forms of protozoa in fæces and can pass them from the gut as early as five minutes and as late as twenty hours after feeding.

(6) Wild flies captured in Alexandria often deposit in their droppings cysts of protozoa and eggs of worms which they have evidently taken up from human dejecta on which they have fed.

(7) A series of weighing experiments show that a single house-fly will take up one milligram of fæces in half an hour.

(8) Cysts of *E. histolytica* will survive in water but are killed instantaneously by drying. The cysts are killed at once by 1 in 20 cresol solution.

DESCRIPTION OF PLATES.

Plate I.

Figs. 1 to 9.—Cysts of *Entamœba histolytica* from three cases to show how distinct strains occur.

1 to 3: Cysts from case Healy, with a strain of *E. histolytica* producing large cysts. 4 to 6: Cysts from case Flynn, with cysts of intermediate sizes. 7 to 9: Cysts from case Russell with cysts of small size. The cysts are drawn to the scale below fig. 9.

Figs. 10 to 23.—*Entamœba nana*, n. sp.

10 and 11: Free forms as seen in fresh preparations. 12 to 17: Free forms as seen in films fixed in Schaudinn's fluid and stained with iron hæmatoxylin. Fig. 15 shows a type of nucleus which is often seen in the encysted forms. 18 to 23: Various stages in the development of the cyst. Forms with one, two, and four nuclei. Fig. 20 shows dividing nuclei. Fig. 22 shows a very characteristic type of nucleus, in which a large chromatin mass on one side of the nuclear membrane is connected by a filament to a small granule on the membrane opposite to it. The free amœbæ measure 5 to 10 microns in diameter, while the cysts are 8 to 10 microns in length.

Plate II.

Figs. 1 to 5.—Various stages in the longitudinal division of *Lamblia intestinalis*.

1: Division of nuclei. 2: Form with four nuclei. 3: Commencing division of body. 4: Final stage of division. 5: Form with two sucking discs developed.

Figs. 6 to 15: *Tetramitus mesnili*.

6 to 9: Forms showing twisting of the body, a condition very commonly seen. 10 to 12: Encysted forms as seen in fresh preparations. The only contents to be seen are a few refractile granules. 13 to 15: Encysted forms stained with iron hæmatoxylin. The nucleus, cytostome with its flagellum and some granules are clearly brought out. Size of cysts, 7 to 10 microns in length.

Figs. 16 to 18.—A yeast which in shape and size closely simulates the cysts of tetramitus.

Plate III.

Figs. 1 to 11.—*Tricercomonas intestinalis*, n. g., n. sp.

1 to 4: Flagellate as seen in the living condition with three anterior flagella and flattened side along which runs the fourth flagellum to become free at the posterior extremity. 5 to 8: Cysts which are probably those of the flagellate with which they were associated. 9 to 11: Flagellates as seen in stained films. The flagellates are 4 to 8 microns in diameter; the cysts 6 to 8 microns in length.

Figs. 12 to 17.—Iodine cysts as seen in stained films. The iodophilic body appears as a vacuole. Often there is a difficulty in distinguishing cysts of *E. histolytica*. The nucleus however is smaller than that of the *E. histolytica* cyst with single nucleus. Fig. 15: I-cyst without vacuoles. The cysts shown in the figures measure 7 to 10 microns in longest diameter.

Figs. 18 to 23.—*Trichomonas intestinalis*.

18: Pentatrichomonas form. 19 to 23: Tetratrichomonas forms. 18 to 20 are from osmic vapour preparations, stained by eosin azur. 21 to 23 are from sublimate wet fixed films stained by iron hæmatoxylin.

Plate IV.

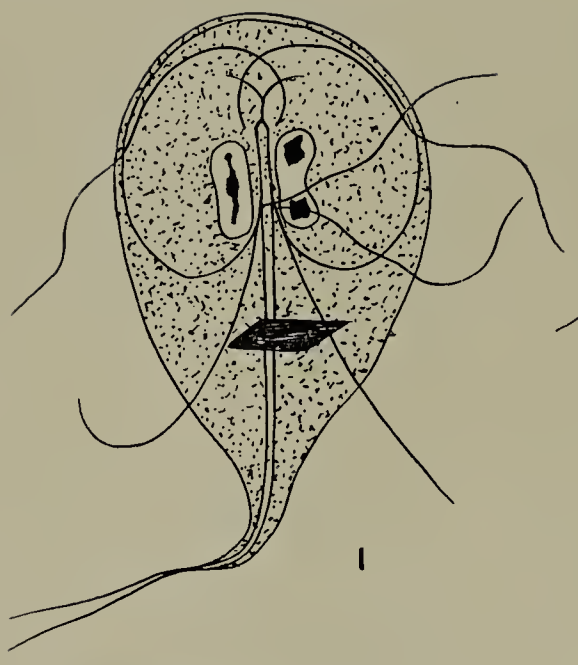
Figs. 1 to 20.—*Waskia intestinalis*, n. g., n. sp.

1 to 8: Flagellates as seen in fresh preparations; 1: form with characteristic bird-like appearance; 2, 4, 6 forms showing cytostome with the thick flagellum projecting from the cytostome and the single thin anterior flagellum. 3 and 5: Dividing forms. 7 and 8: Structureless cysts of flagellate as seen in living condition. 9 to 14: Flagellates as seen in films fixed in sublimate and stained by iron hæmatoxylin. 9 and 13: Dividing forms. 10, 11, 12, 14: Ordinary type of flagellate. 15 to 20: Encysted forms as seen in films fixed in sublimate and stained by iron hæmatoxylin. Some of the cysts show nuclear changes, which may be indications of nuclear divisions. 19: End view of cyst. The length of the free flagellates is 4 to 9 microns, that of the cysts from 4 to 6 microns.

PLATE I.

See page 148.

PLATE II.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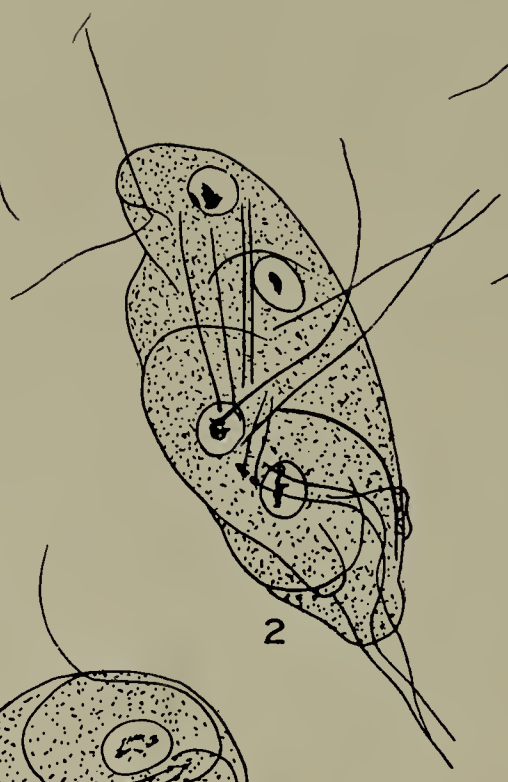
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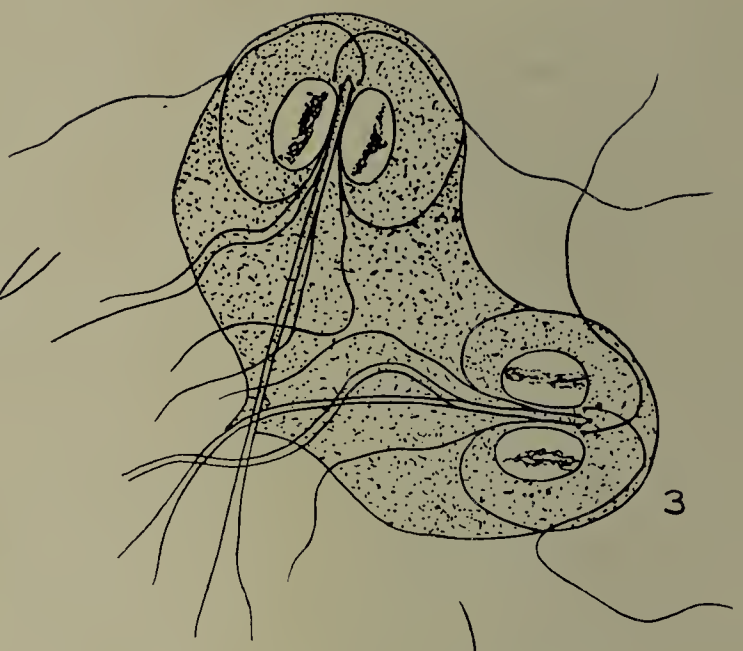
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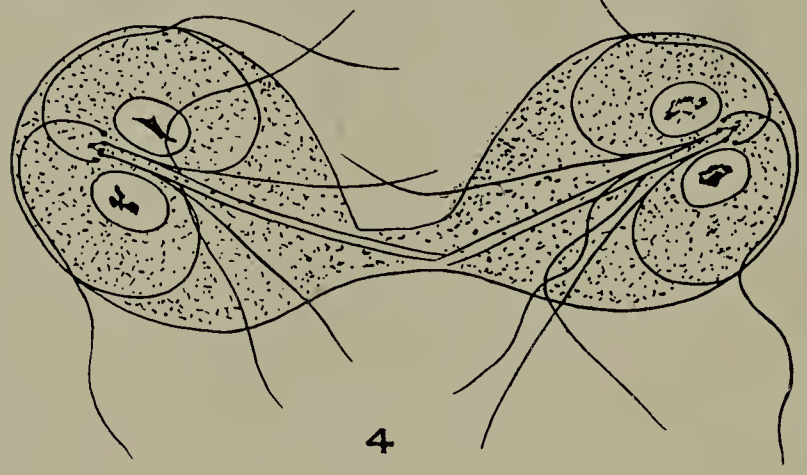
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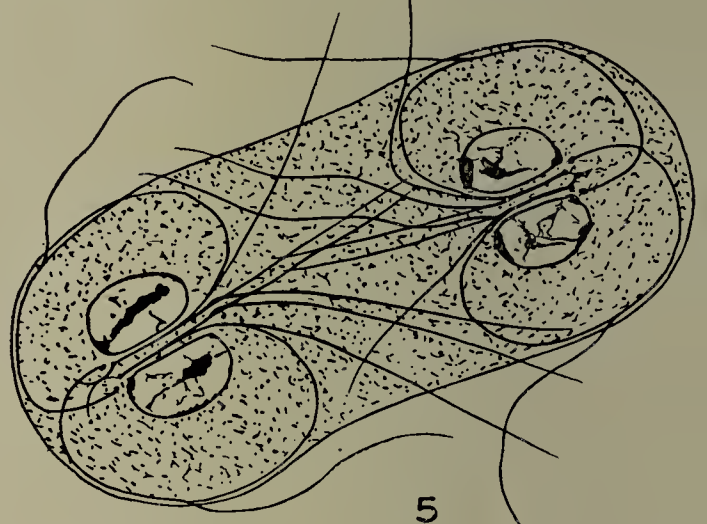
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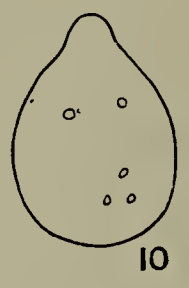
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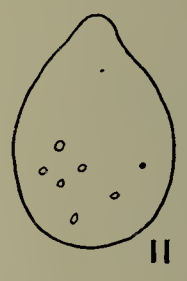
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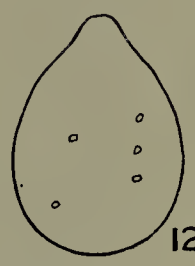
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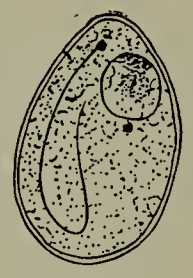
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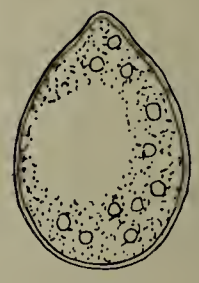
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See page 148.

PLATE III.

See page 148.

PLATE IV.


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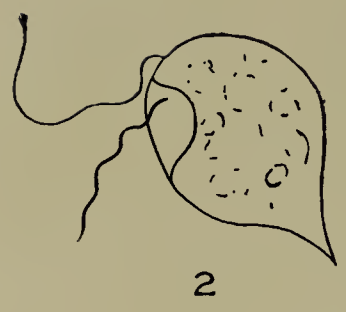
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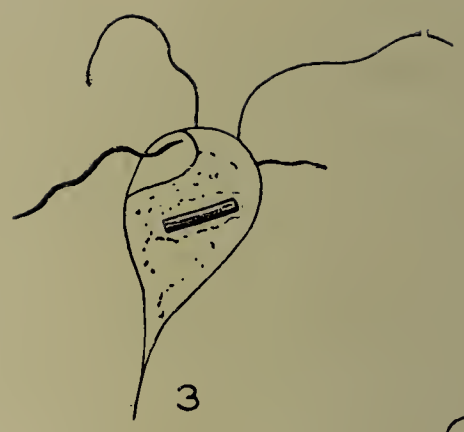
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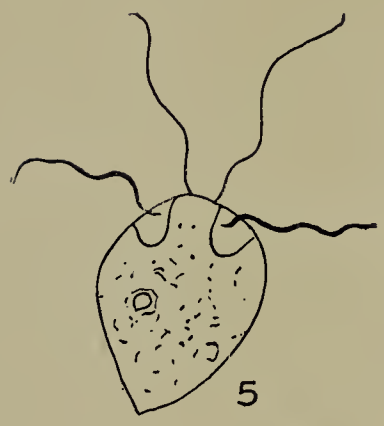
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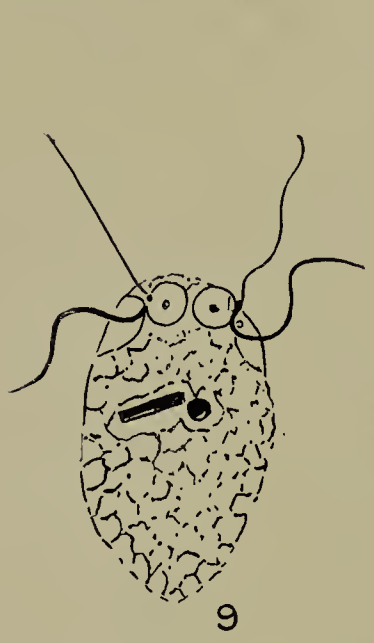
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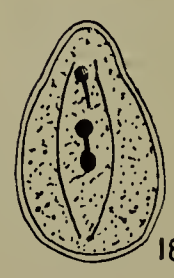
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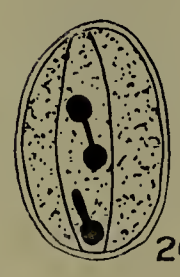
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See page 148.

* THE CARRIAGE OF CYSTS OF *ENTAMŒBA HISTOLYTICA* AND OTHER INTESTINAL PROTOZOA AND EGGS OF PARASITIC WORMS BY HOUSE-FLIES, WITH SOME NOTES ON THE RESISTANCE OF CYSTS TO DISINFECTANTS AND OTHER AGENTS.[1]

THE importance of house-flies in the spread of various bacillary infections of the intestine has been well established and though a similar distribution of the infective agent in amœbic dysentery has been suspected no very definite experiments have been made to prove it. For this reason we feel that the observations to be recorded below are of considerable importance, for they establish beyond dispute the great danger of the house-fly as a factor in the spread of the disease.

Amœbic dysentery is caused by an amœba, *Entamœba histolytica*, which lives in the large intestine, where it invades the wall of the bowel and produces the dysenteric ulceration.

In the acute dysenteric process only the free motile amœbæ are to be found in the fæces, but as the acute symptoms abate smaller amœbæ (minuta forms) occur and many of these become encysted in the large intestine in transparent capsules, in which condition they are passed from the intestine in the fæces in very large numbers. The cysts of *E. histolytica* measure from six to eighteen microns in diameter. On account of the capsule, they are relatively hardy structures which, though they cannot withstand drying, will nevertheless survive for considerable periods if they remain moist. The spread of amœbic dysentery is determined by these cysts, for if ingested in water or food each one gives rise to four amœbæ under the influence of the pancreatic fluid. The four small amœbæ grow into adult forms of *E. histolytica*, which invade the tissues of the large intestine and produce amœbic dysentery.

The cysts are often passed in very large numbers by "carriers," which are cases which have partially or apparently wholly recovered

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[1] Memorandum published in Egypt, April, 1916.

* Reprinted from the *Journal of the Royal Army Medical Corps*, May, 1917.

from amœbic dysentery either after inadequate or ineffective treatment or after no treatment at all. If the cysts from such cases find their way into the water supply or into moist food without having been dried they are likely to give rise to outbreaks of amœbic dysentery, so that any agent like a fly which brings about their distribution must be regarded with suspicion.

(A) Experiments with House-flies (Musca and Fannia).

(1) *Do the Flies ingest the Cysts?*—Kuenen and Swellengrebel (*Cent. of Bakt.*, Bd. 71), working in Sumatra, showed that flies ingested cysts of *E. histolytica* when they fed on infected fæces. This point we readily confirmed by allowing flies to feed on fæces containing cysts and dissecting the flies shortly after.* We found in every case that the gut contained fæces and that in this fæcal material in all parts of the intestine of the fly the cysts occurred just as they had done in the specimen of fæces on which the flies had fed. We were able to demonstrate also that the flies ingested in a similar manner the larger cysts (fifteen to twenty-five microns) of the non-pathogenic human entamœba, *E. coli*, and the cysts of the flagellate *Lamblia intestinalis*.

(2) *Survival of the Cysts in the Intestine of the Fly.*—Kuenen and Swellengrebel stated that the cysts quickly degenerated in the fly's gut and soon became unrecognizable. We dissected flies at various intervals after feeding and noted that so long as fæces were present in the gut cysts could be found, but that they vanished with the disappearance of the fæces from the flies' intestines. Cysts of *E. histolytica*, *E. coli* and *Lamblia intestinalis* were all found in the intestine twenty-four hours after the last feed on fæces. After this time the flies have generally emptied their gut of fæces and then no cysts could be discovered. In one instance cysts of *E. coli* were found in the gut so long as forty-two hours after the last feed. The question of the vitality of these cysts is discussed below, but it may be remarked here that from their normal and living appearance after their sojourn in the fly there can be no doubt that the majority were still alive and therefore infective. Furthermore we have observed the passage through the intestine of the fly of living and active Trichomonas. The application of the eosin test, to be described below, also lent support to this view.

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* Captain J. G. Thompson, R.A.M.C., informed us that he had made a similar observation that flies were able to ingest the cysts.

(3) *How do the Cysts escape from the Fly ?*—We may say at once that we have not yet been able to demonstrate that flies regurgitate through the proboscis cysts they have previously ingested. In the fæces passed by the flies soon after feeding the cysts are readily found.* Within twenty to thirty minutes of feeding on human fæces, the flies begin to deposit droplets of liquid fæces and in these it is easy to detect the unaltered and living cysts. If the fly has continuous access to fæces it will feed every few minutes and as often evacuate its intestinal contents. The amount of material passed through the gut of a single fly in this way must be considerable and within a few hours many thousands of cysts must have followed this course. The cysts, however, may remain in the gut of the fly for some hours and be deposited later, as the following experiment demonstrates. A batch of flies was fed upon fæces containing cysts of *E. histolytica*. After they had fed, the fæces were removed and the flies left without food for sixteen hours. They were then given sugar and water upon which they fed greedily. Shortly after this, they passed droplets of fæces, and in these typical unaltered cysts were found. It is thus evident that flies which have ingested cysts will retain them for considerable periods only to deposit them later upon anything which appeals to their varied tastes. The experiments described were conducted with small numbers of flies, yet there was no difficulty in recovering the infective amœbic cysts in the numerous droplets of fæces they passed. When one reflects on the myriads of flies which swarm about the latrines or fæces deposited in the open in hot countries, one can only be surprised that amœbic dysentery is not more widespread than is actually the case. In these countries fæces, especially when liquid, are devoured and transported *in toto* by these insects only to be deposited broadcast in millions of cyst-infected fæcal droplets upon all kinds of human food, which appears to occupy as an article of diet only a second place in the estimation of these dipterous pests.

It might be urged that cysts could be transported by the adherence of moist fæces to the legs, proboscis and body of the fly. Observations on this question were made by Kuenen and Swellengrebel, who, like Nicol in his work on the passage of worm ova through flies, came to the conclusion that these insects when fouled by fæces did not move far till they had perfectly cleaned themselves.

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* We explained our results and methods to Captain J. G. Thompson, R.A.M.C., who subsequently was able to repeat and confirm some of our observations on the escape of cysts from the fly.

In so doing the flies removed most of the fæces, the remainder drying so that all the cysts were killed. The above-mentioned observers (Kuenen and Swellengrebel) failed to demonstrate the passage of cysts through the intestine and so concluded erroneously that flies were of little consequence in the distribution of the cysts of *E. histolytica*. The experiments we have made entirely disprove this assumption, for with the rapid passage of ingested cysts through the intestine the fly becomes a very potent factor in the spread of amœbic dysentery.

These experiments have been conducted with the ordinary house-flies (Musca and Fannia), and with the blue-bottle fly (Calliphora), and the green-bottle fly (Lucilia). We have observed the passage through all of these of the cysts of *E. coli*, *E. histolytica* and *L. intestinalis*. Quite recently we have examined 200 wild house-flies captured at random in different localities in Alexandria. The flies were given no food whatever by us, but were allowed to deposit their fæces in glass tubes. It was evident that many of these had been feeding on human fæces, and in the droppings of fifteen we found not only the cysts of *E. histolytica*, *E. coli* and *L. intestinalis*, but also the oöcyst of a coccidium and the eggs of various parasitic worms (*Tænia saginata*, *Ankylostoma duodenale*, *Trichocephalus trichiurus*, *Heterophyes heterophyes*, and the comparatively enormous lateral-spined egg of Bilharzia). One fly which deposited cysts of *E. histolytica* was actually captured in a cook-house. It is evident, therefore, that flies under natural conditions are actively concerned in the carriage of the cysts of the dysentery amœbæ and other organisms. Of the 200 flies the droppings of which were examined, fifteen were found to have deposited cysts of protozoa or eggs of parasitic worms. All the infected flies came from near the cook-house of a hospital compound which was separated from a native village by a single wall.

(B) Experiments on the Resistance of Cysts.

The great difficulty in studying the resistance of the cysts is the want of a reliable test as to their viability. Kuenen and Swellengrebel employed the eosin test, which seems to us fairly trustworthy. It is generally agreed that a living cell will not stain with dilute eosin, whereas a dead cell will stain at once. This test can be readily illustrated by the action of heat on the cyst of *E. coli*, *E. histolytica* and *L. intestinalis*. Fæces containing

these cysts when mixed with dilute eosin show under the microscope a red background of stained debris while the white unstained cysts stand out clearly. A few of the cysts, however, may stain with eosin and these are probably dead ones. If the fæces mixed with a little water is heated to boiling point for a second it will be found that after this treatment the eosin will stain all the cysts a deep red instantaneously. In this case there can be little doubt that the cysts have been killed by heat. Drying the cysts has a similar effect so far as making them stain with eosin is concerned. With disinfecting agents, the stronger the solution employed, the more quickly do the cysts acquire the property of taking up the stain. Reagents such as strong sublimate solutions which would be expected to kill the cysts instantaneously similarly cause them to stain with eosin. It seems, therefore, clear that the eosin-staining cysts are dead, though it may be argued that others which do not stain may be dead also or, at any rate, non-infective. Still, if we accept the eosin test as a criterion and regard all unstained cysts as living, the error in judgment will be on the safe side. The following experiments have been made :—

(1) *Drying.*—The cysts of *E. histolytica* do not appear to withstand drying, for they stain with eosin at once after this. Kuenen and Swellengrebel likewise found that drying killed the cysts. The test can be applied by simply allowing fæces to dry at laboratory temperature. The dried fæces is emulsified with saline solution or water, when it will be found that the cysts will stain at once if eosin is added.

(2) *Moisture.*—The cysts will survive for over thirty days in water, an observation which confirms the results of Kuenen and Swellengrebel. Apparently the cysts survive best if there is considerable dilution of fæces with water, so that intense bacterial or fungoid overgrowth does not take place.

(3) *Chemical Agents.* — The experiments were conducted by mixing solutions of chemicals of certain strength with equal volumes of emulsion of fæces in water. Small quantities of the mixture were taken out from time to time and tested by the addition of a drop of eosin solution. The staining of the cysts by the eosin occurred practically instantaneously when it took place at all. When some time was required to kill all the cysts it was found that during this period the percentage of stainable cysts gradually increased.

(*A*) *Emetin Hydrochloride.*—The cysts of *E. histolytica* are much more resistant to this drug than are the free amœbæ, for in a strength of 1 in 200 (equal parts of 1 in 100 emetine and fæces emulsion) it failed to kill the cysts even after nine hours' exposure. It has been claimed that a strength of 1 in 100,000 will quickly kill the free amœbæ.

(*B*) *Cresol.*—This reagent killed all cysts immediately in a strength of 1 in 20 (equal parts of 1 in 10 cresol and fæces emulsion), in one minute in a strength of 1 in 30, in half an hour in a strength of 1 in 100, in one hour in a strength of 1 in 200, and not at all in a dilution of 1 in 2,000.

(*C*) *Carbolic Acid.*—The cysts were all killed in fifteen minutes by 1 in 40 carbolic acid, and in seven hours by 1 in 100, while a 1 in 200 solution failed to kill all the cysts in eight and a half hours.

(*D*) *Formalin.*—The cysts were exposed to 1 in 100 formalin. Even after four hours, they did not stain with eosin, though they were very much shrunken and distorted and giving every appearance of having been killed.

(*E*) *Acid Sodium Sulphate.*—This drug in tablet form as used for the purification of water had no action on the cysts.

(*F*) *Chlorinated Lime Tabloids* (*B. W. & Co.*).—This reagent as used for water sterilization had no action on the cysts. The tabloid gives an equivalent of one grain (0·065 gramme) of chlorine per ten gallons of water (1 in 700,000).*

From the foregoing observations it may be concluded that the cysts of *E. histolytica* are fairly resistant structures, but are quickly killed if deprived of moisture. They certainly will not withstand the desiccation of a tropical sun, so that it seems improbable that wind in blowing about dust can play an important part in their spread. Wind, however, may distribute moist particles of fæces or fragments only externally dry or even portions adhering to pieces of paper or leaves. Of the few reagents we have tried cresol seems the best and would be effective in a strength of 1 in 40 or 50. It is important that the disinfectant should have access to the cysts, and to this end the fæces must be intimately mixed with the fluid added to it. Cresol can, therefore, be employed safely for the disinfection of dysenteric stools, or the hands of those who have to deal with

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* In a test subsequently carried out it was found that free chlorine in water at a strength of 1 in 10,000 failed to kill the cysts after several hours' exposure.

patients. Flies must be of very great importance in the spread of amœbic dysentery, and the results recorded above afford another argument, if, indeed, any further argument is needed, in favour of unceasing warfare against these noxious pests. It may be impossible to isolate and cure every carrier case in a large body of men, but much can be done by the careful use of fly-proof latrines and covered receptacles. With an efficient system of fly and fæces destruction and arrangements for the prevention of flies coming into contact with excreta, there is every reason to believe that amœbic dysentery as well as many other intestinal disorders would be very materially reduced, if not entirely eradicated.

APPENDIX.

PART V.

CHARTS GIVING THE HISTORY OF CASES TREATED FOR *E. HISTOLYTICA* OR FLAGELLATE INFECTIONS.

The cases treated have already been referred to in Part III of this report, where the main results have been indicated. The following charts have been selected to illustrate in detail the course of the various infections. In each instance a short history of the case is given, and this is followed by a tabular arrangement showing the days on which the stool was examined, the course of treatment, the various infections present, and the character of the stool. By this method it is possible to see at a glance if the treatment is having any action on the protozoa. The extent of each infection is indicated by the signs +, ++, or +++. The tables bring out clearly the intermittent character of the various protozoal infections.

During the observations on the cases which were treated the stools were examined, if possible, every day while the patient was in hospital, and afterwards, if he went to convalescent camp (indicated by a C in the first column of the chart), on alternate days. In the second column the treatment is inserted opposite the days on which it was given. In some cases two, or even three, courses of treatment were given when relapses occurred.

Emetin administered by the mouth (E.m.) was frequently followed by vomiting. The occasions on which this happened are shown by a V, followed by the interval which had elapsed after taking the drug.

The column marked E.f. shows free amœbæ, which are left undiagnosed, for when *E. histolytica* cysts and *E. coli* cysts were both present it was evident the associated free amœbæ might belong to either of these.

The cases are arranged in five Sections:—

Section I gives the charts of some of the cases which were treated with one-grain injections, and which have been tabulated in Table X. Some of these cases had more than one treatment, so that, as already explained, they appear in other Tables also.

Cases Harris, Jackson, A. Main, Ball and Barrie, when treated by injections (E.), had already been treated with emetin by the mouth (E.m.) and had relapsed. They appear in Tables X and XI.

Cases Webber, Pero, McQuade, Squires, Obbard, Dorter, Smith and Rushforth relapsed after emetin injections, and were given later the combined treatment of one-grain emetin injection with $\frac{1}{2}$ grain by the mouth (E. 1 E.m. $\frac{1}{2}$). They appear in Tables X and XII.

Case Bennett relapsed after the emetin injections and was then cured by emetin by the mouth. His case appears in Tables X and XI.

Cases Spiers, Healy, Kettlewell and Gaskin all relapsed after emetin injections and were later treated by emetin by the mouth. They appear in Tables X and XI.

Section II gives charts of some of the cases which were treated by orally administered emetin (Table XI). Other cases treated by this method, and which had had also treatment by emetin injections, are included in *Section I.*

Section III gives charts of cases which were treated by the combined method of emetin injections and orally administered emetin (Table XII). Other relapsing cases afterwards treated by this method and which had had previous treatment by emetin injections alone (E.), or emetin by the mouth alone (E.m.), appear in *Section I* or *Section II.*

The action of methyl emetin sulphate is illustrated by the charts of three of the four cases which were treated with this drug. They also received courses of emetin hydrochloride and are included in (*Section I*) Smith, (*Section II*) Blair, and (*Section III*) Russel, H.

Section IV includes chart of one of three cases (McCaffrey, Huntly and Hirst) which illustrates how an *E. histolytica* infection may apparently disappear, at any rate for a considerable period, without any treatment whatever. Case Hirst is especially interesting, for there appeared a *Waskia intestinalis* infection, apparently contracted from case Morris while he was under observation in hospital.

Section V includes cases of flagellate infections which were treated with emetin, β-naphthol or bismuth salicylate. One of these cases developed an *E. histolytica* infection during the course of treatment of the flagellate infection.

ABBREVIATIONS.

In the charts of the cases the following abbreviations have been used for the infections. If the stool was not examined on any day during the course of treatment, a dash (—) appears in the stool column :—

E.h.c. = *E. histolytica* cyst.
E.c.c. = *E. coli* cyst.
E.f. = Unencysted entamœbæ (either *E. histolytica* or *E. coli*).
E.f. r.b.c. = Unencysted entamœbæ with included red blood corpuscles (*E. histolytica*).
L.c. = Lamblia cyst.
L.f. = Unencysted lamblia.
E.n.c. = *E. nana* cyst.
E.n.f. = Unencysted *E. nana*.
Tet.c. = Tetramitus cyst.
Tet.f. = Unencysted tetramitus.
Trich. = Trichomonas.
W.c. = Waskia cyst.
W.f. = Unencysted waskia.
Tc. = Tricercomonas.

The abbreviations used for the character of the stools more or less explain themselves. The first part of the description refers to the colour of the stool and the second to the consistency :—

B. = Brown.
L.b. = Light brown.
Y. = Yellow.
Y.w. = Yellowish white.
F. = Formed.
Sf. = Semiformed.
Uf. = Unformed.
Bk. = Black.
D.g. = Dark green.
D.b. = Dark brown.
Sl. = Slate.
L. = Liquid.
M. = Mucus.
B.m. = Blood and mucus.

Thus L.b.uf. m. means a light brown unformed stool with mucus. B.uf. b.m. means a brown unformed stool with blood and mucus.

In the column on treatment the following are used :—

E. = Emetin hydrochloride injection (E.1 = one grain injection).
E.m. = Emetin hydrochloride by the mouth (E.m.½ = half a grain by the mouth).
E.1 E.m.½ = One grain emetin hydrochloride injection and half a grain by the mouth.
M.E. = Methyl emetin sulphate injection.
M.E.m. = Methyl emetin sulphate by the mouth.
Thy. = Thymol.
P. ipecac. = Pulv. ipecac.
β-n. = β-naphthol.
B.s. = Bismuth salicylate.
Turp. = Turpentine.
V. = Occasions on which vomiting occurred after orally administered emetin. The interval of time before this occurred is inserted.
C. = In first column is the day on which the patient went to the convalescent camp.

Section I.

Case Ure, aged 20.—Patient, who had never been abroad before, left England on May 24, 1915, and went to Gallipoli (Cape Hellas). He suffered there from jaundice (October, 1915), and later had dysentery, for which he had eight injections of emetin (dose ?). He was invalided to Alexandria and admitted to hospital as enteric (December 15, 1915). The stool was examined on February 6, 1916, and found to contain cysts and free *minuta* forms of *E. histolytica*. He received emetin injections of one grain a day from February 21 to March 3. The cysts and amœbæ disappeared by the fifth day of treatment and did not recur. During treatment patient was not confined to bed and had chicken diet. The emetin injections had no effect on the pulse-rate in spite of a previous shortness of breath and an aortic systolic bruit. The temperature remained normal or slightly subnormal. During the last three weeks of control the patient was under observation in the convalescent camp, where he performed light duty.

Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool
		E.h.c.	E.f.				E.h.c.	E.f.				E.h.c.	E.f.	
1	..	+	+	B.sf.	22	E.1	–	–	B.sf.	34	..	–	–	B.sf.
10	..	+++	–	B.sf.	23	E.1	–	–	B.l.	35	..	–	–	B.sf.
12	..	+++	–	B.f.	24	E.1	–	–	B.l.	36	..	–	–	B.sf.
13	..	+++	+++	B.sf.	25	E.1	–	–	B.l.	38	..	–	–	B.sf.
14	..	+++	+++	B.f.	26	E.1	–	–	B.l.	39 C.	..	–	–	B.sf.
15	..	+++	+++	B.f.	27	E.1	–	–	B.l.	41	..	–	–	B.sf.
16	E.1	–	–	–	28	..	–	–	B.sf.	46	..	–	–	B.sf.
17	E.1	+++	+++	B.sf.	29	..	–	–	B.sf.	52	..	–	–	B.sf.
18	E.1	–	+	B.sf.	30	..	–	–	B.sf.	56	..	–	–	B.sf.
19	E.1	–	–	B.sf.	31	..	–	–	B.sf.	58	..	–	–	B.sf.
20	E.1	–	–	B.sf.	32	..	–	–	B.sf.	62	..	–	–	B.sf.
21	E.1	–	–	B.sf.	33	..	–	–	B.sf.					

Case Thompson, A., aged 21.—Patient left England for the first time in 1915, and was on the Peninsula for nine weeks. He then came to Egypt, where he remained 13 weeks, till he was invalided home for dysentery in September. He returned to Egypt in January, 1916, and was found on April 2, in the course of routine examination of cooks in Sidi Bishr Camp, to be a carrier of *E. histolytica*. He was admitted to hospital and found also to harbour *E. coli*, Tetramitus and Lamblia. He was kept under observation till March 11, when a course of emetin (one grain a day for 12 days) was commenced. During treatment he was not kept in bed and was on chicken diet. The treatment abolished the *E. histolytica* infection but not the others. There was no alteration in the temperature or pulse-rate as a result of the emetin. There was no return of the *E. histolytica* infection during a control of one month in the convalescent camp, where patient was on light duty.

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	L.c.	L.f.	Tet.c.	Tet.f.	
1	..	+	+	–	–	–	–	–	B.f.
3	..	+++	–	–	+++	–	–	–	B.f.
4	..	+++	–	–	+++	–	–	–	B.uf.
5	..	++	–	–	+++	–	–	–	B.uf.
6	..	++	+	–	+++	–	–	–	B.l.
7	..	++	++	–	+++	–	–	+	B.uf.
8	..	+++	++	–	+++	–	–	+	B.l.
9	..	++	–	–	+++	++	++	–	B.uf.
10	E.1	++	–	–	+++	–	++	++	B.uf.
11	E.1	–	–	–	–	–	–	–	—
12	E.1	++	++	+	+++	–	++	+++	B.uf.

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	L.c.	L.f.	Tet.c.	Tet.f.	
13	E.1	++	++	+	+++	–	++	+++	B.uf.
14	E.1	–	+++	+	+++	–	++	+++	B.uf.
15	E.1	–	+	–	+++	–	+	–	B.uf.
16	E.1	–	–	+	++	–	+	–	L.b.uf.
17	E.1	–	+	–	–	–	–	+++	B.uf.
18	E.1	–	+	–	–	–	++	–	B.sf.
19	E.1	–	–	–	–	–	–	–	B.uf.
20	E.1	–	++	–	–	–	+++	+	B.uf.
21	E.1	–	++	–	–	–	+++	–	B.sf.
22	..	–	–	–	–	–	++	–	B.uf.
23	..	–	–	–	–	–	+++	+++	B.uf.
29	..	–	+	–	+++	–	+++	–	B.f.
31	..	–	–	–	+++	–	++	–	B.f.
33	..	–	–	–	+++	+	–	+	B.l.
36	..	–	–	–	++	–	+++	–	B.uf.
37	..	–	–	+	+++	–	+++	+++	B.uf.
39	..	–	–	–	+++	–	++	+	B.uf.
41	..	–	+++	–	+++	–	+++	–	B.f.
43	..	–	+	+++	+++	–	+	+++	B.f.
45	..	–	–	+	+++	–	+	+++	B.f.
47	..	–	–	+	+++	–	+	+++	B.f.
49	..	–	+	–	+++	–	+++	–	B.f.
51	..	–	++	–	+++	–	+++	–	B.f.
55	..	–	++	–	+++	–	++	–	B.f.

Case Jones, W., aged 48.—Patient, who had not been abroad before, left England on September 12, 1915, for Gallipoli. Patient had diarrhœa with blood and mucus from September 30 to October 7. At the end of the year he went to Mudros and thence to Alexandria. During the course of routine examination of men in Metras Camp he was found on February 19 to be passing *E. histolytica* cysts in large numbers. He had also a small infection of *E. coli*. He was admitted to hospital as a carrier and was given a course of emetin (one grain a day for 12 days) from March 2 to 13. The *E. histolytica* disappeared on the third day and did not recur during the one month's control. During treatment the patient was not confined to bed and had chicken diet. The temperature was normal or subnormal, and there was no increase in the pulse-rate. During the last three weeks of observation the patient was in the convalescent camp performing light duty.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	+++	+	–	B.f.	19	E.1	–	–	–	B.uf.
4	..	–	–	–	B.l.	20	E.1	–	–	–	B.l.
5	..	–	–	–	B.l.	21	E.1	–	+	–	B.sf.
6	..	+++	+	–	B.sf.	22	E.1	–	–	–	—
7	..	+++	–	+	B.f.	23	E.1	–	–	–	B.sf.
8	..	+++	++	–	B.f.	24	E.1	–	–	–	B.uf
9	..	++	–	+	B.f.	25	..	–	–	–	B.uf.
10	..	+++	–	–	B.sf.	26 C.	..	–	–	–	B.uf.
11	..	+++	–	++	B.uf.	31	..	–	+++	–	B.uf.
12	..	+	–	–	B.f.	35	..	–	++	+	B.uf.
13	E.1	+++	–	–	B.uf.	38	..	–	–	–	B.uf.
14	E.1	–	–	++	B.l.	43	..	–	++	–	B.f.
15	E.1	–	–	+	B.uf.	45	..	–	+	–	B.f.
16	E.1	–	–	–	B.l.	49	..	–	++	–	B.f.
17	E.1	–	–	–	B.uf.	52	..	–	+	–	B.f.
18	E.1	–	–	–	B.uf.	56	..	–	–	–	B.f.

Case Wynne, aged 33.—Patient, who had never been abroad before, went to Gallipoli (Cape Hellas) in May, 1915. After being one week there he contracted dysentery and was ill

for one week. He returned to duty and was transferred to Suvla in August, where he again had dysentery. He remained on the Peninsula till the evacuation, when he came to Egypt. Here he again had an attack of dysentery from which he recovered. He says he was never given emetin injections. On February 13, 1916, during the course of routine examination of men in Mustapha Camp, he was found to be passing large numbers of *E. histolytica* cysts. He was kept under observation till February 23, when a course of emetin injections (one grain a day for 12 days) was commenced. The infection disappeared after five days and did not recur during the one month's control after treatment. During the last three weeks of control patient was in the convalescent camp, where he performed light duty. During treatment he was not kept in bed and was on chicken diet. The treatment had no influence on the temperature or pulse-rate. Tetramitus was present but the infection disappeared during the treatment and did not recur.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.f.	Tet.c.	Tet.f.	
1	..	+++	++	–	–	B.sf.	22	..	–	–	–	–	B.l.m.
3	..	+++	–	–	–	B.uf.	23	..	–	–	–	–	B.uf.
4	..	–	+++	–	+++	B.l.m.	24	..	–	–	–	–	B.uf.
6	..	–	–	–	–	B.f.	25	..	–	–	–	–	B.uf.
7	..	+++	–	–	–	B.l.	26	..	–	–	–	–	B.l.
8	..	+	–	–	–	B.sf.	27	..	–	–	–	–	B.f.
9	..	+++	–	–	–	B.uf.	28	..	–	–	–	–	B.uf.
10	E.1	+++	–	–	–	B.sf.	29	..	–	–	–	–	B.uf.
11	E.1	–	+++	–	++	B.l.	30	..	–	–	–	–	B.uf.
12	E.1	+	–	–	–	B.sf.	31	..	–	–	–	–	B.l.m.
13	E.1	+	–	–	–	B.sf.	32C.	..	–	–	–	–	B.sf.
14	E.1	+	+	–	–	B.sf.	34	..	–	–	–	–	B.l.m.
15	E.1	++	–	+++	–	B.sf.	37	..	–	–	–	–	B.sf.
16	E.1	–	–	–	–	B.l.	41	..	–	–	–	–	B.f.
17	E.1	–	–	–	+++	B.l.	44	..	–	–	–	–	B.f.
18	E.1	–	–	–	+	B.l.	49	..	–	–	–	–	B.f.
19	E.1	–	–	–	–	B.uf.	51	..	–	–	–	–	B.f.
20	E.1	–	–	–	–	B.l.	55	..	–	–	–	–	B.f.
21	E.1	–	–	–	–	B.uf.							

Case Osgood, C., aged 32.—Patient, who had previously been in the West Indies, left England in August, 1915, and went to Mudros where he stayed six weeks. He was then on the Peninsula and had dysentery there. No emetin was given. He was transferred to Egypt and was on the western Egyptian frontier up to March 8, 1916. He returned to Alexandria, and on April 4 was found to be a carrier of *E. histolytica* during the routine examination of cooks in Sidi Bishr Camp. Patient, who had also an infection of *E. nana*, was kept under observation till April 25, when he was given a course of emetin injections of one grain a day for 12 days. The *E. histolytica* quickly disappeared, and during the course there appeared an infection of tetramitus. Patient was controlled for a month after treatment (the last three weeks in the convalescent camp), and there was no recurrence of the *E. histolytica* infection. During treatment patient was not kept in bed and was given chicken diet. The emetin had no effect on the temperature or pulse-rate.

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	E.n.c.	E.n.f.	
1	..	++	++	–	–	–	–	–	B.uf.
3	..	–	–	+	–	–	+	–	B.uf.
4	..	–	–	–	–	–	+	–	B.f.
6	..	–	–	–	–	–	++	++	B.uf.
7	..	–	–	–	–	–	++	++	B.uf.
8	..	–	–	–	–	–	++	–	B.uf.
9	..	+	–	+	–	–	+	+++	B.l.

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	E.n.c.	E.n.f.	
10	..	+	—	—	—	—	—	+++	B.uf.
11	..	—	—	—	—	—	—	+++	B.uf.
12	E.1	+	—	—	+	++	—	—	B.uf.
13	E.1	—	—	+	—	—	—	+++	B.uf.
14	E.1	—	—	—	++	++	—	++	B.uf.
15	E.1	—	—	—	++	+++	—	—	B.uf.
16	E.1	—	—	—	+++	—	—	—	B.uf.
17	E.1	—	—	—	—	++	—	+	B.uf.
18	E.1	—	—	—	++	++	+++	+++	B.sf.
19	E.1	—	—	—	+++	—	—	++	B.uf.
20	E.1	—	—	—	+++	—	—	++	B.uf.
21	E.1	—	—	—	+++	+++	—	—	B.uf.
22	E.1	—	—	—	—	—	—	—	—
23	E.1	—	—	—	+++	+++	—	—	B.uf.
24	..	—	—	—	+++	—	—	—	B.uf.
26 C.	..	—	—	—	—	—	—	—	B.f.
30	..	—	—	—	—	—	+++	+++	B.f.
32	..	—	—	—	+	+	—	—	B.f.
34	..	—	—	—	+++	—	—	—	B.f.
36	..	—	—	—	—	—	+	—	B.f.
38	..	—	—	—	—	—	++	++	B.f.
40	..	—	—	—	—	—	++	++	B.f.
44	..	—	—	—	—	—	++	++	B.f.
46	..	—	—	—	—	—	--	—	B.sf.
48	..	—	—	—	—	—	++	++	B.uf.

Case Turnbull, J., aged 32.—Patient, who had never been abroad before, left England in May, 1915, and was on the Peninsula for seven months. He had an attack of dysentery while there, and was given several injections of emetin. Patient came to Egypt at end of 1915, and on April 20, 1916, was found to be passing mucus in the stool and amœbæ, which were thought to be *E. histolytica*, during the routine examination of cooks in Mustapha Camp. There was present also an infection of *E. coli*, and later a tetramitus appeared. On April 25 *E. histolytica* cysts were found. Patient was given a course of emetin injections (one grain a day for 12 days) from April 26. The *E. histolytica* infection disappeared after four doses of emetin and did not recur during a control of one month, the last 10 days of which patient spent in the convalescent camp on light duty. During treatment patient was not kept in bed and was given chicken diet. The treatment had no effect on the temperature or pulse-rate.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	
1	..	—	—	+++	—	—	B.uf.m.	21	..	—	—	++	+	+++	B.l.
2	..	—	+	+++	—	—	B.uf.m.	23 C.	..	—	++	+	+++	+++	B.f.
5	..	—	—	—	—	—	B.uf.	25	..	—	+	++	+	+++	B.sf.
6	E.1	++	+	—	—	—	B.uf.	27	..	—	—	+	—	+++	B.uf.
7	E.1	+++	—	—	—	—	B.uf.	29	..	—	—	—	—	+++	B.uf.
8	E.1	++	—	—	—	—	B.uf.	31	..	—	—	—	—	+++	B.uf.
9	E.1	—	—	+	—	—	B.l.	33	..	—	+	++	—	++	L.b.uf.
10	E.1	—	—	—	—	—	B.l.	35	..	—	++	+	++	++	L.b.uf.
11	E.1	—	—	—	—	—	B.l.	37	..	—	+	—	—	++	B.uf.
12	E.1	—	—	—	—	—	B.uf.	39	..	—	+	—	+	+++	B.uf.
13	E.1	—	—	+	+++	+++	B.uf.	41	..	—	+	—	—	++	B.uf.
14	E.1	—	—	—	—	+++	B.uf.	43	..	—	+	++	—	+++	B.sf.
15	E.1	—	—	—	—	++	B.sf.	45	..	—	+	+	—	+	B.sf.
16	E.1	—	—	—	—	+++	B.uf.	47	..	—	—	+	—	+	B.sf.
17	E.1	—	—	—	—	++	B.uf.m.	49	..	—	+	—	—	++	B.l.
18	..	—	—	—	+	+++	B.l.								

Case Morris, J., aged 38.—Patient, who had previously been in India, South Africa and Japan, left England on June 7, 1915. He went to the Peninsula, where he was wounded after five days. He had diarrhœa and traces of blood and mucus. He came to Egypt in July and was employed as cook in Sidi Bishr Camp, where his stool was examined on March 31, 1916, in the course of routine examination of cooks in this camp. There were present in the stool numerous small round and oval cysts which contained four nuclei and were taken to be small forms of *E. histolytica* cysts and in addition small typical cysts of *E. histolytica*. The former subsequently turned out to be cysts of *E. nana*. The patient also had tetramitus and the hitherto undescribed flagellate *Waskia intestinalis*. Patient was given a course of emetin injections (one grain a day for 12 days) from March 13 onwards, but though this abolished his infection for the time being, they all returned later with the exception of the *E. histolytica*. Patient had a very bad pyorrhœa, and examination of the mouth soon after the completion of the course of emetin showed in the pus from the teeth the usual mixture of bacteria — spirochætes, leptothrix, and pus cells, but no amœbæ. There was present constantly a trichomonas infection, and later amœbæ appeared in large numbers. The constant presence of trichomonas in the mouth and their equally constant absence from the stool is of interest from the point of view of the difference in species of the mouth and intestinal forms.

Days	Treatment	Findings									Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Tet.c.	Tet.f.	W.c.	W.f.	
1	..	++	—	—	++	—	—	—	—	—	B.f.
3	..	—	—	—	+++	—	—	—	—	—	B.sf.
4	..	+	—	—	+++	—	—	—	—	—	B.l.
5	..	—	—	—	++	—	—	—	—	—	B.l.
8	..	++	—	—	++	—	—	—	+	+++	B.l.
9	..	++	—	—	++	+++	—	—	+++	++	B.l.
11	..	—	—	—	++	—	—	—	+++	—	B.uf.
13	..	—	—	—	++	+++	—	—	+++	—	B.uf.
14	E.1	+	—	++	++	++	—	—	++	+++	B.l.
15	E.1	++	—	—	++	++	—	—	++	—	B.uf.
16	E.1	—	—	+	—	++	—	—	—	+++	B.l.
17	E.1	—	—	—	—	+++	—	—	+++	—	B.uf.
18	E.1	—	—	—	—	—	—	—	—	—	—
19	E.1	—	—	—	—	++	—	—	—	—	L.b.l.
20	E.1	—	—	—	—	—	—	—	—	—	B.uf.
21	E.1	—	—	—	—	—	—	—	—	—	B.uf.
22	E.1	—	—	—	—	—	—	—	—	—	B.uf.
23	E.1	—	—	—	—	—	—	—	—	—	B.l.
24	E.1	+	—	—	—	—	—	—	++	+++	B.l.
25	E.1	—	+	+	—	—	—	—	+++	+++	B.l.
26	..	—	—	—	—	++	—	—	+++	+++	B.l.
27	..	—	—	—	+++	+++	—	—	+++	+++	B.l.
28	..	—	—	—	+++	+++	—	—	+++	+++	B.l.
29	..	—	+	—	++	++	—	—	+++	+++	B.l.
30	..	—	+	—	—	+++	—	—	++	—	B.l.
31	..	—	—	—	—	+++	—	—	—	+++	B.l.
32	..	—	—	—	++	++	—	—	—	++	B.uf.
33	..	—	—	—	—	++	—	—	++	—	B.uf.
34	..	—	—	—	—	++	—	—	++	—	B.l.
35	..	—	—	—	—	+++	—	—	+++	—	B.l.
36	..	—	—	+	—	+++	—	—	+++	—	B.uf.
37	..	—	+	—	++	++	—	—	++	+++	B.l.
38	..	—	—	++	++	++	—	—	+++	—	B.uf.
39	..	—	—	—	++	++	—	—	—	++	B.l.
40	..	—	—	+	—	+++	—	—	—	—	B.l.
41	..	—	—	++	—	+++	—	—	+++	+++	B.l.
42	..	—	+	++	+++	+++	—	+	++	—	B.l.

Days	Treatment	Findings									Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Tet.c.	Tet.f.	W.c.	W.f.	
43	..	—	—	++	—	+++	—	++	—	+++	B.l.
44	..	—	—	++	—	+++	—	++	—	+++	B.l.
45	..	—	—	—	—	+++	—	++	—	+++	L.bl.
46	..	—	—	++	—	+++	—	+++	++	+++	B.l.
47	..	—	—	+	—	+++	—	+	+	+	B.l.
49	..	—	—	+	+++	—	—	++	—	+	B.l.
50	..	—	—	—	—	+++	—	+	++	++	B.l.
53	..	—	—	—	—	—	—	—	—	+	B.f.
54	..	—	—	—	—	+	—	—	—	—	B.l.
55	..	—	—	—	++	++	—	—	—	—	B.l.
56	..	—	+	++	+++	+++	++	++	—	—	B.f.
57	..	—	+	—	++	++	++	—	—	—	B.uf.
58	..	—	—	—	+++	+++	+++	—	—	—	B.uf.
59	..	—	+	+	+++	+++	++	++	—	—	B.l.
60	..	—	++	+	+	+	++	++	—	—	B.uf.
61	..	—	+	—	++	++	—	—	—	—	B.l.
64	..	—	—	—	++	++	—	—	—	—	B.uf.

Case Wood, J., aged 35.—Patient, who had never been abroad before, left England in June, 1915, and went direct to the Peninsula, where he remained for 6½ months. While there he had an attack of dysentery (blood and mucus) but had no emetin. He came to Egypt in December, 1916. On March 27, during the routine examination of men in Mustapha Camp he was found to be a carrier of *E. histolytica*. Patient had also trichomonas and *E. nana* infections. On March 31 and April 1 one grain of emetin by the mouth was given. This caused the patient to vomit at once on both occasions, so on April 2 a course of emetin injections (one grain a day for 12 days) was commenced. The *E. histolytica* disappeared from the stool after the second dose of emetin by the mouth in spite of the vomiting. There was no recurrence of the infection. The trichomonas and *E. nana* infection disappeared during the treatment, but recurred later. During treatment the patient was not kept in bed and was on chicken diet. The emetin had no action on the temperature or pulse-rate.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.f.	E.n.c.	E.n.f.	Trich.				E.h.c.	E.f.	E.n.c.	E.n.f.	Trich.	
1	..	++	++	—	—	—	B.sf.	21	..	—	—	—	—	+++	B.l.
3	..	—	+++	—	—	+++	B.l.	22	..	—	—	—	+	+++	B.uf.
4	..	+++	—	+++	—	+++	B.uf.	23	..	—	—	++	++	++	B.uf.
5	E.m.1. V. inst.	+++	—	+++	+++	+++	B.uf.	24	..	—	—	+++	+++	++	B.uf.
								25	..	—	—	+++	—	—	B.f.
6	E.m.1. V. inst.	+++	—	+++	—	—	B.uf.	26	..	—	—	+++	—	—	B.uf.
								27	..	—	—	+++	—	—	B.l.
7	E.1	—	—	+++	—	—	L.b.f.	28	..	—	—	+++	+++	—	B.uf.
8	E.1	—	—	—	—	—	B.sf.	29	..	—	—	—	—	—	B.uf.
9	E.1	—	—	—	—	—	B.sf.	30	..	—	—	—	—	—	B.uf.
10	E.1	—	—	+++	+++	—	B.sf.	31	..	—	—	—	+	—	B.uf.
11	E.1	—	—	—	—	—	B.sf.	32	..	—	—	—	+++	—	B.uf.
12	E.1	—	—	—	—	—	B.l.	33	..	—	—	—	+++	—	B.uf.
13	E.1	—	—	—	—	—	B.uf.	34	..	—	—	—	+	—	B.uf.
14	E.1	—	—	—	—	—	B.sf.	35	..	—	—	++	++	—	B.uf.
15	E.1	—	—	—	—	—	B.uf.	36	..	—	—	—	—	—	B.uf.
16	E.1	—	—	—	—	—	B.uf.	37	..	—	—	++	++	—	B.uf.
17	E.1	—	—	—	—	—	B.uf.	38C.	..	—	—	++	++	+	B.uf.
18	E.1	—	—	—	—	—	B.l.	41	..	—	—	++	++	—	B.f.
19	..	—	—	—	—	—	B.l.	45	..	—	—	++	++	+	B.f.

Case Harris, C., aged 25.—Patient, who had been in France from January to August, 1915, was transferred to the Peninsula, where he remained four months. Patient states that he had dysentery both in France and on the Peninsula, and that he had had at one time or another 25 injections of emetin. He came to Egypt early in 1916, and was found to be a carrier of *E. histolytica* on March 22 during the routine examination of men in Mustapha

Camp. He had also infections of *E. coli* and I-cysts, while later, infections of trichomonas and tetramitus appeared. He was kept under observation till March 28, when a course of emetin by the mouth (one grain a day for 12 days) was commenced. Patient took this emetin without vomiting, with the result that the *E. histolytica*, *E. coli*, and I-cyst infections disappeared after three days. The *E. coli*, however, reappeared on the last day of the course, and the *E. histolytica* and trichomonas within a week. From April 14 to 25 patient was given emetin injections (one grain a day for 12 days). The *E. histolytica* infection again disappeared after four injections, but not the *E. coli* nor the trichomonas, which had reappeared before the injections were commenced. Tetramitus was also found for the first time towards the end of the course of injections. During treatment patient was not kept in bed and was on chicken diet. He was kept under control for one month in the convalescent camp, where he performed light duty. There was no recurrence of the *E. histolytica* infection, while the two emetin courses had no effect on the temperature or pulse-rate.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	Trich.	
1	..	++	+++	++	—	—	—	B.f.
3	..	—	+	—	—	—	—	B.f.
4	..	—	—	—	—	—	—	B.uf.
5	..	+	—	—	—	—	—	B.f.
6	..	++	+	—	—	—	—	B.f.
7	E.m.1	++	++	—	—	—	+	B.uf.
8	E.m.1	++	+	—	—	—	—	B.uf.
9	E.m.1	++	+	—	—	—	—	B.f.
10	E.m.1	—	+	+	—	—	—	B.uf.
11	E.m.1	—	—	—	—	—	—	B.f.
12	E.m.1	—	—	—	—	—	—	B.f.
13	E.m.1	—	—	—	—	—	—	B.f.
14	E.m.1	—	—	—	—	—	—	B.sf.
15	E.m.1	—	—	—	—	—	—	B.sf.
16	E.m.1	—	—	—	—	—	—	B.uf.
17	E.m.1	—	—	—	—	—	—	B.uf.
18	E.m.1	—	++	—	—	—	—	B.uf.
19	..	—	—	—	—	—	—	B.uf.
20	..	—	++	—	—	—	—	B.uf.
21	..	—	++	—	—	—	++	B.l.
22	..	—	+	—	—	—	++	B.uf.
23	..	—	+	—	—	—	++	B.sf.
24	..	—	+	—	—	—	—	B.sf.
25	E.1	++	++	+	—	—	++	B.uf.
26	E.1	+	++	—	—	—	—	B.uf.
27	E.1	+	+	—	—	—	+++	B.uf.
28	E.1	—	++	—	—	—	++	B.sf.
29	E.1	—	++	+	—	—	++	B.uf.
30	E.1	—	+	+	—	—	++	B.uf.
31	E.1	—	++	—	—	—	—	B.uf.
32	E.1	—	++	—	—	—	+	B.uf.
33	E.1	—	—	+	—	+	++	B.l.
34	E.1	—	—	—	—	+	+++	B.uf.
35	E.1	—	—	—	—	—	++	B.uf.
36	E.1	—	—	—	—	+++	—	B.l.
37 C.	..	—	+	—	++	+++	—	B.uf.
40	..	—	+++	—	+++	+++	—	B.uf.
42	..	—	+	—	—	++	—	B.f.
45	..	—	++	—	—	—	—	B.f.
47	..	—	++	+	—	—	—	B.f.
49	..	—	++	++	—	—	—	B.f.
51	..	—	+	+	—	—	+++	B.f.
53	..	—	++	+	—	—	—	B.f.
55	..	—	++	+	—	—	—	B.f.
57	..	—	+	+	—	—	—	B.f.
59	..	—	—	+	—	—	—	B.f.
61	..	—	+	—	—	—	—	B.f.
63	..	—	—	—	—	—	—	B.f.
65	..	—	—	—	—	—	—	B.f.

Case Russell, F., aged 35.—Patient, who had never been abroad before, left England in August, 1915, for the Peninsula, where he remained three months. He came to Egypt in December, 1915, and on March 21, 1916, was found to be a carrier of *E. histolytica* during the routine examination of cooks at Sidi Bishr Camp. Patient had dysentery whilst on the Peninsula, but had no emetin. There was present also an I-cyst infection, which disappeared before treatment was commenced on April 11. The cysts of *E. histolytica* in this case were very small, having an average diameter of 7 to 10 microns. From April 11 to 22 patient was given emetin injections of one grain a day for 12 days. The *E. histolytica* disappeared after the second dose and did not recur during a control of one month, which patient spent in the convalescent camp, where he performed light duty. After a week's stay in the camp patient contracted an infection of *E. nana*. During treatment he was not kept in bed and was on chicken diet. The emetin had no effect on the temperature, but the pulse-rate was definitely accelerated towards the end of the course.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	E.n.c.	E.n.f.				E.h.c.	E.f.	E.n.c.	E.n.f.	
1	..	+	+++	—	—	B.f.	21	E.1	—	—	—	—	B.uf.
3	..	—	—	—	—	B.f.	22	E.1	—	—	—	—	B.uf.
4	..	—	—	—	—	B.uf.	23	E.1	—	—	—	—	B.uf.
5	..	—	—	—	—	B.f.	24 C.	..	—	—	—	—	B.uf.
6	..	—	—	—	—	B.f.	30	..	—	—	—	—	B.f.
7	..	+	—	—	—	B.f.	32	..	—	—	—	—	B.f.
8	..	—	+++	—	—	B.uf.	34	..	—	—	+++	+++	B.sf.
9	..	+++	+	—	—	B.sf.	36	..	—	—	+++	+++	B.f.
10	..	—	—	—	—	B.uf.	38	..	—	—	+++	+++	B.f.
11	..	+++	+++	—	—	B.uf.	39	..	—	—	++	++	L.b.f.
12	E.1	+++	+++	—	—	B.uf.	40	..	—	—	+	+	B.f.
13	E.1	+++	+++	—	—	B.sf.	42	..	—	—	—	—	B.f.
14	E.1	—	—	—	—	B.uf.	44	..	—	—	+	+	B.f.
15	E.1	—	—	—	—	B.uf.	46	..	—	—	+	+	B.f.
16	E.1	—	—	—	—	B.uf.	48	..	—	—	+	+	B.f.
17	E.1	—	—	—	—	B.uf.	50	..	—	—	+	+	B.f.
18	E.1	—	—	—	—	B.uf.	52	..	—	—	+	+	B.f.
19	E.1	—	—	—	—	B.f.	54	..	—	—	+++	+++	B.f.
20	E.1	—	—	—	—	B.uf.							

Case Myers M., aged (?).—Patient, who had not been abroad before, left England in October, 1915, and came direct to Egypt. On April 20, 1916, he was found to be a carrier of *E. histolytica*. During the routine examination of cooks in Mustapha Camp, he had also an infection of I-cysts, and later on there appeared infections of trichomonas, tetramitus, *E. coli*, *E. nana*, and on one occasion only tricercomonas. There was no history of dysentery. Patient was given a course of emetin injections (one grain a day for 12 days) from April 25 onwards. The *E. histolytica* infection disappeared and did not recur during a control of one month, the last three weeks of which patient spent in the convalescent camp, where he performed light duty. The emetin had no effect on the patient's temperature or pulse-rate. During treatment he was not kept in bed and was on chicken diet.

Days	Treatment	Findings									Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Tet.c.	Tet.f.	Trich.	T.c.	
1	..	+++	—	—	—	—	—	—	—	—	B.uf.
3	..	++	—	++	—	—	—	—	—	—	B.l.
4	..	++	—	—	—	—	—	—	—	—	B.uf.
5	..	—	—	—	—	—	—	—	—	—	B.uf.
6	E.1	—	—	—	—	—	—	—	—	—	B.uf.
7	E.1	++	—	—	—	—	—	—	—	—	B.uf.
8	E.1	—	—	—	—	—	—	—	—	—	B.uf.

Days	Treatment	Findings									Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Tet.c.	Tet.f.	Trich.	T.c.	
9	E.1	—	—	+	—	—	—	++	—	—	B.uf.
10	E.1	—	—	—	—	—	—	—	—	—	B.l.
11	E.1	—	—	—	—	—	—	—	—	—	B.l.
12	E.1	—	—	++	—	—	—	—	+++	—	B.l.
13	E.1	—	—	—	—	—	++	—	—	—	B.l.
14	E.1	—	—	—	—	—	+	—	++	—	B.l.
15	E.1	—	—	—	—	—	—	—	+++	—	B.sf.
16	E.1	—	—	—	—	—	—	+++	++	—	B.sf.
17	E.1	—	+	+	—	—	—	++	++	—	B.l.
18	..	—	—	—	—	—	—	—	+++	—	B.l.
21	..	—	—	—	—	—	—	—	—	—	B.uf.
23 C.	..	—	—	—	—	—	—	++	++	—	B.f.
25	..	—	—	—	—	—	+	—	—	—	B.f.
27	..	—	—	—	—	—	—	++	—	—	B.f.
29	..	—	—	—	+++	+++	+	—	+	—	B.f.
31	..	—	++	++	+++	+++	—	—	—	—	B.f.
33	..	—	++	++	++	++	—	—	—	—	B.f.
37	..	—	++	+	—	—	—	—	—	—	B.f.
39	..	—	+	++	—	—	—	—	—	++	B.f.
41	..	—	+	—	—	—	—	—	—	—	B.uf.
43	..	—	+++	—	—	—	—	—	—	—	B.sf.
45	..	—	+++	—	—	—	—	—	—	—	B.sf.
47	..	—	+	—	—	—	—	—	—	—	B.sf.

Case Nicholson, E., aged 43.—Patient, who had seen previous foreign service in South Africa, left England on March 16, 1915, and went to Mudros where he remained five months. He had several attacks of diarrhœa, but no dysentery. He then came to Alexandria, where he again had attacks of diarrhœa. He was found to be passing large numbers of *E. histolytica* cysts on February 15, 1916, in the course of routine examination of men in Camp A (Gabbari). He was kept under observation till March 4, and was then given a course of emetin injections of one grain a day for 12 days. *E. histolytica* was last seen on the eighth day of treatment and the infection did not recur, though the *E. coli* appeared later. During treatment patient was not kept in bed and was on chicken diet. During the last fortnight of control patient was in convalescent camp, where he performed light duty.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	+++	—	—	B.uf.	31	..	—	—	—	B.f.
11	..	—	—	—	B.f.	32	..	—	—	—	B.f.
12	..	—	—	—	B.f.	33	..	—	—	—	B.f.
13	..	—	—	—	B.f.	34	..	—	—	—	B.f.
14	..	+++	—	—	B.uf.	35	..	—	—	—	B.f.
15	..	+++	—	+	B.uf.	36	..	—	—	—	B.uf.
16	..	++	—	—	B.l.	37	..	—	—	—	B.uf.
17	..	+++	—	+++	B.uf.	38	..	—	—	—	B.f.
18	..	++	—	+++	B.l.	39	..	—	—	+	B.uf.
19	E.1	+++	—	—	B.uf.	40	..	—	+	—	B.l.
20	E.1	++	—	+	B.f.	41	..	—	—	—	B.f.
21	E.1	—	—	++	B.uf.	42	..	—	+	—	B.f.
22	E.1	—	—	+++	B.uf.	43	..	—	—	—	B.uf.
23	E.1	—	—	+++	B.l.	44	..	—	—	—	B.uf.
24	E.1	—	—	—	B.sf.	45	..	—	—	—	B.uf.
25	E.1	+	—	—	B.sf.	46 C.	..	—	++	+	B.f.
26	E.1	+	—	—	B.l.	49	..	—	++	—	B.f.
27	E.1	—	—	—	B.sf.	53	..	—	+	—	B.f.
28	E.1	—	—	—	B.l.	57	..	—	—	—	B.f.
29	E.1	—	—	—	B.f.	60	..	—	+	—	B.f.
30	E.1	—	—	—	B.f.						

Case Page, J., aged 27.—Patient, who had never been abroad before, left England for Gallipoli in January, 1915. While there he suffered from diarrhœa but did not have dysentery. He came to Alexandria and was found to be infected with *E. histolytica* on March 30, 1916, in

the routine examination of cooks in Sidi Bishr Camp. He was kept under observation till April 11, when he was given a course of emetin injections (one grain a day for 12 days). The *E. histolytica* infection, as well as a lamblia infection which had appeared, were abolished by the treatment, after which the patient was kept under observation in the convalescent camp for one month. There was no return of the *E. histolytica* infection. The lamblia infection, however, reappeared, and then developed an *E. coli* infection. The emetin treatment had no effect on the temperature or pulse-rate. The patient was kept in bed and was on chicken diet.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.c.c.	E.f.	L.c.				E.h.c.	E.c.c.	E.f.	L.c.	
1	..	++	—	—	—	B.uf.	20	E.1	—	—	—	—	B.f.
3	..	+	—	—	—	B.uf.	21	E.1	—	—	—	—	B.f.
4	..	+++	—	—	—	B.sf.	22	E.1	—	—	—	—	B.f.
5	..	++	—	++	—	B.f.	23	E.1	—	—	—	—	B.f.
6	..	++	—	—	—	B.f.	24	E.1	—	—	—	—	B.uf.
7	..	+	—	—	—	B.f.	25 C.	..	—	—	—	—	B.uf.
8	..	+	—	—	+++	B.f.	33	..	—	—	—	—	B.sf.
9	..	++	—	—	++	B.f.	35	..	—	—	—	—	B.sf.
10	..	+	—	—	++	B.uf.	37	..	—	—	—	—	B.f.
11	..	—	—	—	++	B.uf.	39	..	—	—	—	—	B.f.
12	..	++	—	—	++	B.uf.	41	..	—	—	—	++	B.f.
13	E.1	++	—	—	++	B.f.	43	..	—	—	—	++	B.f.
14	E.1	—	—	—	++	B.f.	45	..	—	—	—	++	B.f.
15	E.1	—	—	—	++	B.uf.	47	..	—	—	—	+	B.f.
16	E.1	—	—	—	+	B.uf.	49	..	—	—	—	—	B.f.
17	E.1	—	—	—	—	B.sf.	51	..	—	—	+	++	B.f.
18	E.1	+	—	—	—	B.sf.	53	..	—	++	++	++	B.f.
19	E.1	+	—	—	—	B.sf.	55	..	—	++	—	++	B.f.

Case Flynn, N., aged 37.—Patient, who had never been abroad before, left England on March 16, 1916, and came direct to Egypt. He had previously been at Woolwich and other parts of England and Ireland. He was found to be a carrier of *E. histolytica* on April 4, 1916, during the routine examination of cooks in Sidi Bishr Camp, and just 10 days after his arrival in Egypt. He had never had dysentery or bad diarrhœa. He was kept under observation till April 13, when a course of emetin injections was commenced (one grain a day for 12 days). The *E. histolytica* cysts did not disappear till after the ninth day of treatment. Patient was kept under control for over a month after treatment, three weeks of which was spent in the convalescent camp. He had infections of *E. coli* and *E. nana*. There was no return of the *E. histolytica* infection. During treatment patient was not kept in bed, and was on chicken diet. The emetin had no effect on patient's temperature or pulse-rate.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.				E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
1	..	+	+	+++	—	—	B.sf.	22	..	—	—	—	+	+	B.uf.
3	..	+++	—	+	—	—	B.uf.	23	..	—	—	—	—	—	B.uf.
4	..	+++	—	+++	—	—	B.uf.	24	..	—	—	+++	++	++	B.uf.
5	..	+++	+	+++	—	—	B.l.	25	..	—	++	—	++	++	B.uf.
6	..	+++	—	++	—	—	L.b.uf.	26	..	—	+	—	++	—	B.uf.
7	..	+++	—	—	—	—	L.b.uf.	27	..	—	—	+	++	+++	B.uf.
9	..	+++	++	—	—	—	B.sf.	28 C.	..	—	+	+	++	++	B.uf.
10	E.1	+++	++	—	—	—	L.b.uf.	32	..	—	—	—	++	++	B.uf.
11	E.1	+++	+	—	—	—	L.b.sf.	34	..	—	+	—	++	++	B.f.
12	E.1	+++	+	—	—	—	B.f.	36	..	—	—	+	—	—	B.f.
13	E.1	+++	+	+++	—	—	B.uf.	37	..	—	—	+	+++	+++	B.f.
14	E.1	—	—	—	—	—	—	40	..	—	—	+	—	—	B.f.
15	E.1	+	++	—	—	—	B.uf.	42	..	—	—	—	—	—	B.f.
16	E.1	—	—	—	—	—	—	44	..	—	—	—	+	+	B.f.
17	E.1	—	—	—	—	—	—	46	..	—	+	—	++	++	B.f.
18	E.1	+++	—	+	—	—	B.uf.	48	..	—	+	+	++	++	B.f.
19	E.1	—	+	—	—	—	B.sf.	50	..	—	++	—	++	++	B.f.
20	E.1	—	—	—	—	—	—	54	..	—	—	—	+	+	B.f.
21	E.1	—	+	—	+	+	B.uf.								

Case Kitson, aged 22.—Patient, who had not been abroad before, left England in April, 1915, for the Peninsula, where he remained 4½ months. He was transferred to Egypt in September, 1915, and on April 22, 1916, was found to be a carrier of *E. histolytica* during the routine examination of men in Mustapha Camp. He was kept under observation till April 26, when a course of emetin injections was commenced (one grain a day for 12 days). The *E. histolytica* infection quickly disappeared, and did not recur during a control of one month, which patient spent in the convalescent camp on light duty. During treatment he was not kept in bed, and was on chicken diet. The emetin had no effect on the patient's temperature or pulse-rate. An accompanying *E. coli* infection was not abolished by the treatment. There had been no history of dysentery.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.				E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
1	..	+++	++	—	—	—	B.uf.	19	..	—	+	+	—	—	B.f.
3	..	+++	++	+	—	—	B.uf.	21	..	—	—	—	—	—	B.f.
4	E.1	+++	++	+++	—	—	B.uf.	23	..	—	++	++	—	—	B.f.
5	E.1	++	++	—	—	—	B.uf.	25	..	—	+	—	+	+	B.f.
6	E.1	—	++	—	—	—	B.uf.	27	..	—	++	—	+	+	B.f.
7	E.1	—	—	—	—	—	B.uf.	31	..	—	+	+	+	+	B.uf.
8	E.1	—	—	—	—	—	B.uf.	33	..	—	—	—	—	—	B.f.
9	E.1	—	—	—	—	—	B.uf.	35	..	—	++	—	—	—	B.f.
10	E.1	—	—	—	—	—	B.l.	37	..	—	+	—	—	—	B.sf.
11	E.1	—	—	—	—	—	—	39	..	—	+++	+	—	—	B.uf.
12	E.1	—	++	—	—	—	B.uf.	41	..	—	+	—	—	—	B.sf.
13	E.1	—	—	—	—	—	B.l.	43	..	—	+	—	—	—	B.sf.
14	E.1	—	—	—	—	—	B.l.	45	..	—	+	—	—	—	B.uf.
15	E.1	—	+	+	—	—	L.b.uf.	47	..	—	+	—	+++	+++	B.sf.
16 C.	..	—	—	—	—	—	L.b.sf.								

Case Knight, aged 21.—Patient, who had been in India a year previously, left England on October 8, 1915, and was on the Peninsula for two months before being transferred to Egypt. Patient, who had never had dysentery, was found to be a carrier of *E. histolytica* on April 17, 1916, during the routine examination of men in Mustapha Camp. He was kept under observation till April 25, when a course of emetin injections (one grain a day for 12 days) was commenced. The *E. histolytica* cysts had disappeared from the stool two days before treatment was commenced, but on the first day of treatment a small number of free entamœbæ were present. There was no recurrence of the infection during a control of one month, the greater part of which patient spent in the convalescent camp, where he performed light duty. The course of emetin had no effect on the pulse-rate, but there was a rise in temperature to 100° on two successive days. During treatment patient was not kept in bed and was on chicken diet.

Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool
		E.h.c.	E.f.				E.h.c.	E.f.				E.h.c.	E.f.	
1	..	++	—	B.uf.	14	E.1	—	—	B.l.	29	..	—	—	B.f.
3	..	+	—	B.f.	15	E.1	—	—	—	31	..	—	—	B.f.
4	..	+	—	B.uf.	16	E.1	—	—	B.uf.	33	..	—	—	B.f.
6	..	+++	—	B.sf.	17	E.1	—	—	—	35	..	—	—	B f.
7	..	—	++	B.l.	18	E.1	—	—	B.l.	39	..	—	—	B.f.
8	..	—	—	B.uf.	19	E.1	—	—	B.uf.	41	..	—	—	B.f.
9	E.1	—	+	B.uf.m.	20	E.1	—	—	B.l.	43	..	—	—	B.uf.
10	E.1	—	—	—	21	..	—	—	B.l.	46	..	—	—	B.f.
11	E.1	—	—	B.uf.	22C.	..	—	—	B.uf.	48	..	—	—	B.f.
12	E.1	—	—	B.uf.	25	..	—	—	B.uf.	50	..	—	—	B.f.
13	E.1	—	—	B.l.	27	..	—	—	B.uf.					

Case Ormrod, T. H., aged 27.—Patient, who had not been abroad before, left England in the middle of 1915 for Mudros, where he remained for five months. He then came to Egypt and was found on February 12, 1916, to be a carrier of *E. histolytica* in the routine examination of men in Camp A. Patient had suffered from attacks of diarrhœa but had not had dysentery. He was kept under observation till February 27, when he was given a course of emetin injections of one grain a day for 12 days. The *E. histolytica* disappeared after the second dose. There was a concurrent *E. coli* infection. There was no recurrence of the *E. histolytica* during a control of one month, the last three weeks of which patient spent in the convalescent camp on light duty. During treatment he was not kept in bed, and was given chicken diet. The course of emetin had no effect on the temperature, though the pulse-rate, which was decidedly slow before, became more rapid during and after treatment.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	+++	—	+++	B.uf.m.	24	E.1	—	—	—	B.sf.
7	..	—	—	—	B.uf.	25	E.1	—	—	—	B.sf.
9	..	—	—	—	L.b.uf.	26	E.1	—	—	—	B.l.
10	..	—	—	—	B.uf.	27	E.1	—	—	—	B.uf.
11	..	+	—	+	B.f.	28	..	—	—	—	B.uf.
12	..	+++	—	—	B.f.	29	..	—	—	—	B.uf.
13	..	+++	+	+	B.sf.	30	..	—	—	—	B.f.
14	..	+++	+++	—	B.f.	31	..	—	—	+	B.sf.
15	..	+++	+++	+	B.f.	33C.	..	—	—	—	B.f.
16	E.1	+++	+++	—	B.f.	35	..	—	+	—	B.f.
17	E.1	++	++	—	B.uf.	38	..	—	++	++	B.f.
18	E.1	—	+	++	B.l.	42	..	—	+++	+++	B.f.
19	E.1	—	++	—	B.f.	45	..	—	—	+	B.uf.
20	E.1	—	—	—	—	50	..	—	+++	—	B.f.
21	E.1	—	+	—	B.l.	52	..	—	+++	+++	B.f.
22	E.1	—	—	—	B.sf.	56	..	—	++	—	B.f.
23	E.1	—	—	—	B.sf.	59	..	—	++	—	B.f.

Case Cooper, L. J., aged 20.—Patient, who had not been abroad before, came to Egypt from England in January, 1916. He was found to be infected with *E. histolytica* on March 2, 1916, during routine examination of cooks in Sidi Bishr Camp. He was kept under observation till March 11, when a course of emetin injections (one grain a day for 12 days) was commenced. The *E. histolytica* infection disappeared after the first dose of emetin and did not recur in a subsequent control of one month after treatment was completed. The *E. coli*, which was present on the first examination and then absent, reappeared during the course of emetin. After treatment the patient was in the convalescent camp, where he performed light duty. The emetin did not appear to have any influence on the patient's temperature or pulse-rate. Just 20 days after the control of this patient was completed he was re-admitted to hospital for dysentery. Examination showed no amœbæ but the cellular picture of a bacillary infection. The stool was examined microscopically for four days and no signs of the original *E. histolytica* infection could be found. There was no history of previous dysentery.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.				E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
1	..	+	+	—	—	—	B.f.	11	E.1	—	—	—	—	—	B.f.
3	..	+	—	—	—	—	B.f.	12	E.1	—	—	—	—	—	B.f.
4	..	+	—	—	—	—	B.uf.	13	E.1	—	—	—	—	—	B.f.
5	..	+	—	—	—	—	B.uf.	14	E.1	—	—	—	—	—	B.sf.
6	..	+++	—	—	—	—	B.f.	15	E.1	—	—	—	—	—	B.f.
7	..	+++	—	—	—	—	B.uf.	16	E.1	—	—	—	—	—	B.f.
8	..	+++	—	—	—	—	B.uf.	17	E.1	—	—	—	—	—	B.uf.
9	..	+++	—	—	—	—	B.f.	18	E.1	—	—	—	—	—	B.uf.
10	E.1	++	—	—	—	—	B.sf.	19	E.1	—	++	—	—	—	B.uf.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.				E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
20	E.1	—	++	—	—	—	B.uf.	44	..	—	++	+	+++	+++	B.f.
21	E.1	—	+++	—	—	—	B.l.	46	..	—	+	++	+++	+++	B.uf.
22 C.	..	—	+++	—	—	—	B.uf.	48	..	—	+	+	+++	+++	B.uf.
28	..	—	+	—	—	—	B.uf.	50	..	—	+	—	—	+++	B.uf.
30	..	—	+	—	—	—	B.f.	52	..	—	+	+	++	+++	B.uf.
32	..	—	+	—	—	—	B.f.	71	Bacil. Dys.	—	—	—	—	—	B.l. B.m.
34	..	—	+	—	—	—	B.f.								
36	..	—	++	+	—	—	B.f.	72	..	—	—	—	—	—	B.l.
38	..	—	+	+	—	—	B.f.	73	..	—	—	—	—	—	B.l.
40	..	—	+	—	—	—	B.f.	74	..	—	—	—	—	—	B.uf.
42	..	—	++	—	—	—	B.f.								

Case Badham, G., aged 34.—Patient, who had never been abroad before, left England in June, 1915, and came direct to Egypt, where he was placed on the orderly staff at the Orwa-el-Waska Hospital, Alexandria. On February 16, 1916, he was found to be a carrier of *E. histolytica* together with *E. coli* in the routine examination of the hospital staff. He was kept under observation till February 25, when a course of emetin injections (one grain a day for 8 days) was instituted. During treatment patient continued his duties and took his ordinary food. The *E. histolytica* disappeared but recurred later—between 16 and 40 days after treatment was finished. Patient, still on duty and taking ordinary food, was given a course of emetin injections (one grain a day for 12 days). The *E. histolytica* infection again disappeared and this time did not recur during a control of 1½ months after treatment. The patient, who was a nervous individual, continued his duties during treatment without any noticeable discomfort.*

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	++	++	—	B.uf.	61	E.1	++	—	—	B.uf.
4	..	+++	—	—	L.b.sf.	62	E.1	—	—	+	B.uf.
5	..	+	+	++	B.sf.	63	E.1	—	—	+++	B.uf.
6	..	+	—	+	B.sf.	64	E.1	—	+	++	B.uf.
7	..	++	—	+++	L.b.uf.m.	65	E.1	—	—	—	B.uf.
8	..	++	++	++	L.b.uf.	66	E.1	—	+	—	B.uf.
9	..	++	++	+	L.b.uf.	67	E.1	—	—	—	B.uf.
10	E.1	++	++	++	L.b.uf.	68	E.1	—	—	—	B.uf.
11	E.1	+	+	—	L.b.uf.	69	E.1	—	—	—	B.uf.
12	E.1	+	+	+	B.uf.	70	E.1	—	+	—	B.uf.
13	..	—	++	+++	B.uf.	71	..	—	—	—	B.f.
14	E.1	—	+	+	B.uf.	73	..	—	++	—	B.f.
15	E.1	—	+	+	B.uf.	75	..	—	+	—	B.f.
16	E.1	++	++	+	B.uf.	77	..	—	+	+	B.f.
17	E.1	—	++	—	B.uf.	79	..	—	+	—	B.f.
18	E.1	—	—	++	B.uf.	82	..	—	—	—	B.f.
20	..	—	+	++	B.uf.	84	..	—	—	—	B.f.
21	..	—	+	++	B.uf.	87	..	—	—	—	B.f.
23	..	—	++	—	B.uf.	89	..	—	—	++	B.f.
24	..	—	++	+	B.uf.	91	..	—	+	—	B.f.
26	..	—	—	+	B.uf.	93	..	—	—	++	B.f.
27	..	—	—	++	B.uf.	95	..	—	+	—	B.sf.
28	..	—	—	+++	B.uf.	97	..	—	—	—	B.uf.
29	..	—	—	—	B.uf.	98	..	—	+	—	B.uf.
30	..	—	—	+++	B.uf.	100	..	—	—	—	B.f.
33	..	—	—	+++	B.uf.	101	..	—	—	+	B.l.
56	..	++	+	++	B.uf.	107	..	—	—	+	B.f.
58	..	++	—	++	B.uf.	109	..	—	—	—	B.f.
59	E.1	++	+	++	B.uf.	110	..	—	++	+++	B.f.
60	E.1	++	—	—	B.uf.	112	..	—	—	++	B.f.

* Note.—An examination made by Mr. Savage, in Egypt one year later, failed to reveal an *E. histolytica* infection, though *E. coli* was still present.

Case Webber, S., aged 32.—Patient, who had never been abroad before, left England in June, 1915. He went direct to the Peninsula, where he stayed seven weeks, during which time he had a short attack of dysentery for which he was given injections of emetin (dose?). In September he came to Egypt, where he was employed as cook. On April 13, 1916, during the routine examination of cooks at Sidi Bishr Camp he was found to be a carrier of *E. histolytica*. He had also an infection of *E. coli* and was later found to have coccidia (isospora). From April 25, patient was given a 12-day course of emetin injections (one grain a day). The patient was not kept in bed and was on chicken diet. The cysts of *E. histolytica* disappeared from the stool but recurred soon after. The coccidia infection was also unaffected by the treatment. Accordingly patient was given a second 12-day course of emetin from May 15 (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth each night). He was kept in bed on milk diet. He vomited on only one occasion. The *E. histolytica* and the coccidia both disappeared from the stool and did not recur during a control of one month, the greater part of which patient spent in the convalescent camp on light duty, where he seemed to pick up an *E. nana* infection. Neither the temperature nor the pulse-rate were in any way effected by the two courses of emetin. There were no symptoms which could be attributed to the coccidia, for though the patient had also an *E. histolytica* infection he was not ill in any way.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Coc.	
1	..	++	+	—	—	—	—	B.uf.
7	..	++	+	+	—	—	—	B.uf.
8	..	+++	—	—	—	—	—	B.uf.
9	..	+++	—	+++	—	—	—	B.uf.
10	..	+++	—	—	—	—	—	B.uf.
11	..	+++	+	—	—	—	—	B.uf.
12	..	+++	+++	—	—	—	—	B.uf.
13	E.1	+++	+++	—	—	—	+	B.uf.
14	E.1	+++	++	+	—	—	+	B.uf.
15	E.1	++	++	—	—	—	+	B.uf.
16	E.1	+	—	—	—	—	+	B.uf.
17	E.1	++	++	—	—	—	—	B.uf.
18	E.1	—	—	+	—	—	+	B.uf.
19	E.1	++	—	—	—	—	+	B.uf.
20	E.1	+	+	—	—	—	—	B.f.
21	E.1	+	+	—	—	—	++	B.uf.
22	E.1	—	—	—	—	—	+	B.uf.
23	E.1	—	—	—	—	—	+	B.uf.
24	E.1	—	—	—	—	—	+	B.uf.
25	..	+	—	—	—	—	++	B.uf.
26	..	+	—	—	—	—	++	B.sf.
27	..	—	—	—	—	—	+	L.b.uf.
28	..	—	—	—	—	—	+	B.uf.
29	..	+	—	+	—	—	++	B.uf.
30	..	—	—	—	—	—	++	B.uf.
31	..	+	—	—	—	—	++	L.b.uf.
32	..	++	—	—	—	—	++	B.sf.
33	E.1 E.m.½	++	—	—	—	—	++	B.uf.
34	E.1 E.m.½	++	—	—	—	—	++	B.uf.
35	E.1 E.m.½	—	—	+	—	—	++	L.b.uf.
36	E.1 E.m.½	—	—	—	—	—	+	L.b.uf.
37	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
38	E.1 E.m.½	—	—	—	—	—	+	L.b.uf.
39	E.1 E.m.½	—	—	—	—	—	—	B.l.
40	E.1 E.m.½	—	—	+	—	—	—	B.l.
41	E.1 E.m.½	—	—	+	—	—	—	B.uf.
42	E.1 E.m.½	—	—	—	—	—	—	B.f.
43	E.1 E.m.½	—	—	—	—	—	—	B.uf.
44	E.1 E.m.½	—	—	—	—	—	—	B.uf.
46 C.	..	—	—	++	—	—	—	B.uf.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Coc.	
48	..	—	+	—	—	—	—	B.uf.
50	..	—	—	—	—	—	—	B.sf.
52	..	—	—	+	—	—	—	B.sf.
54	..	—	—	—	—	—	—	B.sf.
56	..	—	—	++	—	—	—	B.sf.
58	..	—	—	+	++	++	—	B.sf.
60	..	—	—	—	—	—	—	B.sf.
62	..	—	—	—	—	—	—	B.sf.
64	..	—	—	—	+	+	—	B.sf.
66	..	—	—	++	—	—	—	B.sf.
68	..	—	—	++	—	—	—	B.sf.
70	..	—	—	—	—	—	—	B.sf.
72	..	—	—	—	—	—	—	B.sf.
74	..	—	—	+	—	—	—	B.sf.

Case Pero, aged (?).—Patient, who had previously been abroad before in German W. Africa, left England on December 28, 1915, and came direct to Egypt. He was on the western Egyptian frontier for two months and then returned to Alexandria, where, on April 4, he was found to be a carrier of *E. histolytica* during the routine examination of cooks at Sidi Bishr Camp. He had never suffered from dysentery. There was also an infection of *E. coli* and tetramitus, and on two occasions trichomonas was found. Patient was placed on emetin injections (one grain a day for 12 days) from April 15 to 25. He was not kept in bed and was given chicken diet. The *E. histolytica* infection quickly disappeared but returned on May 2, five days after patient had gone to the convalescent camp. He was re-admitted to hospital and a second course of emetin was commenced for 12 days (one grain injection in the morning and ½ grain in keratin-coated tabloid by the mouth at night). During this course patient was kept in bed and given milk diet. He vomited regularly after taking the emetin at night, so after six nights the ½ grain by the mouth was discontinued. In spite of this all three infections disappeared quickly and during the control of over one month after treatment the tetramitus infection alone reappeared. For the last three weeks of observation patient was in convalescent camp, where he performed light duty. The first course of emetin had no effect on the patient's temperature or pulse-rate, but the second course produced a rapid pulse which persisted for over a week after the course was completed.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	Trich.	
1	..	+	++	—	—	—	—	B.uf.
4	..	+	+	—	—	—	—	B.uf.
5	..	+	+	+	—	—	—	B.uf.
7	..	—	—	—	++	+++	—	B.uf.
8	E.1	+	—	—	++	+++	—	B.uf.
9	E.1	—	—	—	—	—	—	B.l.
10	E.1	—	+	—	+++	—	—	B.uf.
11	E.1	—	—	—	—	—	—	—
12	E.1	+	+	—	+++	—	—	B.uf.
13	E.1	—	—	—	—	—	—	—
14	E.1	—	—	—	—	—	—	—
15	E.1	—	—	++	+	+++	++	B.uf.
16	E.1	—	—	—	+++	—	—	B.sf.
17	E.1	—	—	—	+	+++	—	B.uf.
18	E.1	—	—	—	—	—	—	B.uf.
19	E.1	—	—	—	—	+++	—	B.l.
20	..	—	—	—	—	—	+	B.uf.
21 C.	..	—	—	—	—	+++	—	B.uf.
26	..	+++	—	—	—	—	—	B.uf.
28	..	+++	—	—	—	—	—	B.uf.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	Trich.	
29	E.1	—	—	—	—	—	—	—
	E.m.½. V.½ hr.							
30	E.1	+++	—	—	++	—	—	B.f.
	E.m.½. V.2½ hrs.							
31	E.1	—	—	—	—	—	—	L.b.uf.
	E.m.½. V.1 hr.							
32	E.1	—	—	—	—	—	—	B.l.
	E.m.½. V.½ hr.							
33	E.1	—	—	—	—	—	—	B.uf.
	E.m.½. V.10 min.							
34	E.1	—	—	—	—	—	—	B.uf.
	E.m.½. V.10 min.							
35	E.1	—	—	—	—	—	—	B.uf.
36	E.1	—	—	—	—	—	—	L.b.uf.
37	E.1	—	—	—	—	—	—	L.b.l.
38	E.1	—	—	—	—	—	—	L.b.l.
39	E.1	—	—	—	—	—	—	L.b.uf.
40	E.1	—	—	—	—	—	—	B.uf.
41	..	—	—	—	—	—	—	B.uf.
42	..	—	—	—	—	++	—	B.sf.
44	..	—	—	—	—	++	—	B.l.
45 C.	..	—	—	—	—	+++	—	B.uf.
47	..	—	—	—	++	++	—	B.f.
52	..	—	—	—	—	—	—	B.uf.
58	..	—	—	—	—	—	—	B.sf.
62	..	—	—	—	—	—	—	B.sf.
64	..	—	—	—	—	—	—	B.sf.
66	..	—	—	—	—	—	—	B.sf.
68	..	—	—	—	—	—	—	B.sf.
70	..	—	—	—	—	—	—	B.sf.

Case McQuade, aged 36.—Patient, who had previous foreign service in South and West Africa, where he suffered from malaria, left England on September 16, 1915, for the Peninsula. He was there four months and was then transferred to Egypt, where, on March 23, 1916, he was found to be a carrier of *E. histolytica* during the routine examination of men in Mustapha Camp. Patient says he never suffered from dysentery at any time. He was kept under observation till March 28, when a course of emetin injections of one grain a day for 12 days was commenced. The *E. histolytica* infection disappeared after the second dose, and during the course a tetramitus infection appeared. During treatment patient was not kept in bed and was given chicken diet. Ten days after the course was finished patient went to the convalescent camp and a fortnight later was again passing cysts of *E. histolytica*. He was taken into hospital again and on April 5 another course of emetin was commenced. This time he was given each day for 12 days a one-grain injection of emetin in the morning and ½ grain of emetin in keratin-coated tabloid at night. He was kept in bed on milk diet. The cysts of *E. histolytica* disappeared after the first day and did not recur in a control of one month, the last three weeks of which patient spent in the convalescent camp. The emetin by the mouth produced no vomiting and there was no change in the temperature or pulse-rate.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.f.	Tet.c.	Tet.f.	
1	..	++	—	—	—	B.f.	10	E.1	—	—	—	—	B.uf.
3	..	+++	—	—	—	B.uf.	11	E.1	—	—	++	—	B.uf.
5	..	—	—	—	—	B.sf.	12	E.1	—	—	+++	—	B.f.
6	E.1	+	—	—	—	B.f.	13	E.1	—	—	+	—	B.f.
7	E.1	+	—	—	—	B.uf.	14	E.1	—	—	++	—	B.uf.
8	E.1	—	—	—	—	B.f.	15	E.1	—	—	+	—	B.uf.
9	E.1	—	—	—	—	B.uf.	16	E.1	—	—	—	—	B.l.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.f.	Tet.c.	Tet.f.	
17	E.1	—	—	—	—	B.uf.	51	E.1 E.m.½	—	—	—	—	L.b.l.
18	..	—	—	—	—	B.f.	52	E.1 E.m.½	—	—	—	—	L.b.f.
19	..	—	—	—	—	B.f.	53	E.1 E.m.½	—	—	—	—	L.b.uf.
20	..	—	—	—	—	B.sf.	54	E.1 E.m.½	—	—	—	—	L.b.uf.
21	..	—	—	—	—	B.uf.	55	E.1 E.m.½	—	—	—	—	L.b.uf.
22	..	—	—	—	—	B.f.	56	..	—	—	—	—	L.b.uf.
23	..	—	—	—	—	B.f.	57	..	—	—	—	—	B.sf.
24	..	—	—	—	—	B.f.	59	..	—	—	—	—	B.l.
25	..	—	—	—	—	B.f.	61 C.	..	—	—	—	—	B.f.
30	..	—	—	+	++	B.uf.	64	..	—	—	—	—	B.uf.
33	..	—	—	—	+	B.f.	66	..	—	—	—	—	B.f.
39	..	++	—	—	—	B.f.	68	..	—	—	—	—	B.sf.
41	..	+++	—	—	—	B.f.	70	..	—	—	—	—	B.uf.
43	..	+++	—	—	—	B.f.	72	..	—	—	—	—	B.sf.
44	E.1 E.m.½	++	—	—	—	L.b.f.	74	..	—	—	—	—	B.sf.
45	E.1 E.m.½	—	—	—	—	L.b.l.	76	..	—	—	—	—	B.sf.
46	E.1 E.m.½	—	—	—	—	L.b.sf.	78	..	—	—	—	—	B.sf.
47	E.1 E.m.½	—	—	—	—	L.b.f.	80	..	—	—	—	—	B.sf.
48	E.1 E.m.½	—	—	—	—	—	82	..	—	—	—	—	B.sf.
49	E.1 E.m.½	—	—	—	—	L.b.sf.	84	..	—	—	—	—	B.sf.
50	E.1 E.m.½	—	—	—	—	L.b.sf.	86	..	—	—	—	—	B.sf.

Case Squires, H., aged 32.—Patient, who had not been abroad before, left England in September, 1915, and went to Mudros, where he remained three months before being transferred to Egypt. On April 2, 1916, during the routine examination of cooks in Sidi Bishr Camp he was found to be a carrier of *E. histolytica*. There was no history of previous dysentery. There was also an infection of *E. coli* and *E. nana*, and on one occasion lamblia cysts were present. Patient was kept under observation till April 10, when a course of emetin injections (one grain a day for 12 days) was commenced. During treatment patient was not kept in bed and was given chicken diet. The *E. histolytica* disappeared, but were again present within a week of the completion of the course. Patient, who had gone to the convalescent camp, returned to hospital and was given a second course of emetin for 12 days (one-grain injection each morning and ½ grain in keratin-coated tabloid each night). During the course he was kept in bed on milk diet. There was no vomiting. This treatment had the effect of abolishing all the infections, including a tricercomonas infection which appeared while patient was in a convalescent camp. After the second course of emetin patient was kept under observation for a month, the last three weeks of which were spent in the convalescent camp, where he performed light duty. The *E. coli* and *E. nana* infections recurred, but not the *E. histolytica* nor the tricercomonas infections. Neither course of emetin had any effect on the patient's temperature or pulse-rate.

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	L.c.	T.c.	
1	..	+	+	—	—	—	—	—	B.sf.
4	..	+	—	—	++	++	—	—	B.uf.
5	..	++	—	—	—	—	—	—	B.sf.
6	..	++	—	—	—	—	—	—	B.sf.
7	..	++	++	—	—	—	—	—	B.f.
8	..	+++	++	—	++	—	—	—	B.sf.
9	..	+++	++	—	—	—	—	—	B.f.
10	E.1	++	++	—	++	—	—	—	B.f.
11	E.1	—	+	—	++	—	—	—	B.f.
12	E.1	—	—	—	—	—	—	—	—
13	E.1	—	+	—	—	+++	++	—	B.uf.
14	E.1	—	—	—	—	—	—	—	—

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	L.c.	T.c.	
15	E.1	—	+	—	—	—	—	—	B.uf.
16	E.1	—	++	—	—	—	—	—	B.f.
17	E.1	—	+	—	—	—	—	—	B.uf.
18	E.1	—	+++	—	—	—	—	—	B.f.
19	E.1	—	++	—	—	—	—	—	B.f.
20	E.1	—	—	—	—	—	—	—	—
21	E.1	—	+	—	—	—	—	—	B.uf.
22 C.	..	—	+++	—	—	—	—	—	B.uf.
28	..	—	++	—	++	++	—	—	B.f.
31	..	+	—	—	—	—	—	—	B.f.
32	..	—	—	—	—	—	—	—	B.f.
34	..	—	++	—	++	++	—	—	B.uf.
35	..	—	+	—	++	++	—	+++	B.sf.
36	..	—	+	—	++	++	—	+++	B.sf.
37	E.1 E.m.½	++	—	—	—	—	—	—	B.uf.
38	E.1 E.m.½	+	—	+	++	++	—	+++	B.l.
39	E.1 E.m.½	—	+	+	—	+++	—	++	B.uf.
40	E.1 E.m.½	—	—	—	++	—	—	—	L.b.uf.
41	E.1 E.m.½	—	—	—	—	—	—	—	L.b.l.
42	E.1 E.m.½	—	—	—	—	—	—	—	B.l.
43	E.1 E.m.½	—	—	—	—	—	—	—	B.l.
44	E.1 E.m.½	—	—	—	+++	+++	—	+++	B.uf.
45	E.1 E.m.½	—	—	—	++	++	—	—	L.b.uf.
46	E.1 E.m.½	—	—	—	++	—	—	—	L.b.uf.
47	E.1 E.m.½	—	—	—	—	—	—	—	L.b.sf.
48	E.1 E.m.½	—	—	—	—	—	—	—	L.b.uf.
49	..	—	—	—	—	—	—	—	L.b.uf.
50	..	—	—	—	+++	+++	—	—	B.f.
51	..	—	—	—	++	++	—	—	B.f.
52 C.	..	—	—	—	+++	+++	—	—	B.sf.
54	..	—	+	—	+++	+++	—	—	B.f.
56	..	—	++	—	+++	+++	—	—	B.f.
58	..	—	+++	—	+++	+++	—	—	B.uf.
62	..	—	+	—	+++	+++	—	—	B.f.
64	..	—	+	—	++	++	—	—	B.sf.
66	..	—	++	—	+++	+++	—	—	B.sf.
68	..	—	—	—	—	—	—	—	B.sf.
70	..	—	+	—	+++	+++	—	—	B.sf.
72	..	—	+	—	++	++	—	—	B.sf.
74	..	—	—	—	+	—	—	—	B.sf.
76	..	—	—	—	—	—	—	—	B.sf.
78	..	—	—	—	—	—	—	—	B.sf.
80	..	—	—	—	—	—	—	—	B.sf.

Case Boyd, aged 37.—Patient, who had not been abroad before, left England on March 2, 1916, and came direct to Egypt, where on April 6, during the routine examination of cooks in Sidi Bishr Camp, he was found to be infected with *E. histolytica*. He had also an infection of tetramitus, and later on showed at one time or another infection of *E. coli* and tricercomonas. A lamblia infection appeared at the end of the observation, the patient having probably become infected in camp. There was no history of dysentery. Patient was kept under observation till April 15, when a course of emetin injections was commenced (one grain a day for 12 days). During the course he was not kept in bed and was on chicken diet. The *E. histolytica* cysts disappeared from the stool before the end of the course, but recurred again within a week. Patient, who had gone to the convalescent camp, was re-admitted to hospital, and a second course of emetin given (one-grain injection each morning, and ½ grain in keratin-coated tabloid each night). Patient was kept in bed on milk diet. There was no vomiting. The *E. histolytica* quickly disappeared and did not recur during a control of over one month, during the greater part of which patient spent in the convalescent camp on light duty. The courses of emetin had no effect on the patient's temperature or pulse-rate.

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	L.c.	Tet.c.	Tet.f.	T.c.	
1	..	++	—	+	—	—	—	—	B.uf.
3	..	—	—	—	—	—	—	—	B.l.
4	..	—	—	—	—	—	+++	—	B.sf.
5	..	—	—	—	—	—	++	—	B.uf.
6	..	—	—	—	—	—	++	—	B.uf.
7	..	—	—	—	—	—	—	—	B.uf.
9	..	+++	—	—	—	—	—	—	B.uf.
10	E.1	+++	—	—	—	—	++	—	B.uf.
11	E.1	+	—	++	—	—	—	—	B.l.
12	E.1	—	—	—	—	—	—	—	B.sf.
13	E.1	—	—	—	—	—	—	—	B.uf.
14	E.1	—	—	+	—	—	+	—	B.l.
15	E.1	—	—	—	—	+	—	—	B.f.
16	E.1	++	—	++	—	—	+++	—	B.sf.
17	E.1	++	—	—	—	—	—	—	B.l.
18	E.1	—	—	—	—	—	—	—	B.uf.
19	E.1	—	—	—	—	—	—	—	B.uf.
20	E.1	—	—	—	—	—	—	—	B.uf.
21	E.1	—	—	+	—	—	+	—	B.uf.
22	..	—	—	—	—	—	—	—	B.f.
25	..	—	—	—	—	—	—	—	B.f.
26 C.	..	—	—	—	—	—	—	—	B.f.
29	..	++	+	—	—	++	—	—	B.f.
30	..	++	—	+	—	—	+	—	B.f.
34	..	+	—	—	—	—	+	—	B.f.
37	..	+++	—	+	—	++	+++	++	B.sf.
38	E.1 E.m.½	++	—	+	—	++	+++	+++	B.uf.
39	E.1 E.m.½	+	—	—	—	—	—	—	B.f.
40	E.1 E.m.½	+	—	—	—	—	—	—	B.sf.
41	E.1 E.m.½	—	—	—	—	—	—	—	L.b.sf.
42	E.1 E.m.½	—	—	—	—	—	—	—	L.b.f.
43	E.1 E.m.½	—	—	—	—	—	—	—	B.uf.
44	E.1 E.m.½	—	—	—	—	—	—	—	B.f.
45	E.1 E.m.½	—	—	—	—	—	—	—	B.sf.
46	E.1 E.m.½	—	—	—	—	—	—	—	B.sf.
47	E.1 E.m.½	—	—	—	—	—	—	—	L.b.sf.
48	E.1 E.m.½	—	—	—	—	—	—	—	B.uf.
49	E.1 E.m.½	—	—	—	—	—	—	—	L.b.uf.
51	..	—	—	—	—	—	—	—	B.uf.
52 C.	..	—	—	—	—	—	—	—	B.uf.
53	..	—	—	—	—	—	—	—	B.uf.
55	..	—	—	—	—	—	—	—	B.uf.
57	..	—	—	—	—	—	—	—	B.uf.
59	..	—	—	—	—	—	—	—	B.uf.
63	..	—	—	—	—	—	—	—	B.sf.
65	..	—	—	—	—	—	+++	—	B.sf.
67	..	—	—	—	—	+	+++	—	B.sf.
69	..	—	—	—	—	—	+	—	B.sf.
71	..	—	—	—	—	—	—	—	B.sf.
73	..	—	—	—	—	—	—	—	B.sf.
75	..	—	—	—	—	—	—	—	B.sf.
77	..	—	—	—	—	—	—	—	B.sf.
79	..	—	—	—	—	—	—	—	B.sf.
81	..	—	—	—	++	—	+++	—	B.sf.

Case Cox, J., aged 20.—Patient, who had never been abroad before, left England in May, 1915, and came direct to Egypt, and was stationed at the 19th General Hospital. On February 15, during the routine examination of the Hospital Staff, patient was found to be passing cysts of *E. histolytica* in small numbers. The stool was not examined again till April 17, when cysts were still present but in larger numbers. There was also an infection of *E. coli* and *E. nana*, and trichomonas appeared later. Patient stated that he had never had dysentery or diarrhœa. From April 20 he was given a 12-day course of emetin injections (one grain a day). The cysts of *E. histolytica* were last seen on the fifth day of treatment. During the treatment patient continued his duties in the hospital and was on full diet.

The cysts of *E. histolytica* reappeared in the stool 25 days after the emetin course was completed. The course of emetin had no effect on the patient's general health.*

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Trich.	
1	..	+	—	—	—	—	—	B.uf.
63	..	++	++	+	—	—	—	B.uf.
66	E.1	++	+	—	—	—	—	B.uf.
67	E.1	++	+	—	—	—	—	B.uf.
68	E.1	+++	—	—	—	—	—	B.f.
69	E.1	+	+	—	—	—	—	B.f.
70	E.1	+++	++	—	—	—	—	B.sf.
71	E.1	++	++	—	—	—	—	B.uf.
72	E.1	—	+	—	—	—	—	B.uf.
73	E.1	—	+	—	—	—	—	B.uf.
74	E.1	—	—	—	—	—	—	B.f.
75	E.1	—	—	+	—	—	—	B.f.
76	E.1	—	++	+	—	—	—	B.f.
77	E.1	—	++	+	—	—	—	B.f.
78	..	—	+++	—	—	—	+++	B.uf.
81	..	—	++	—	—	—	++	B.uf.
83	..	—	+	—	—	—	—	B.uf.
86	..	—	+	+	—	—	—	B.uf.
88	..	—	+	—	—	—	—	B.uf.
90	..	—	—	—	—	—	—	B.f.
92	..	—	—	++	—	—	—	B.f.
93	..	—	—	+	+	++	+	B.f.
98	..	—	—	+	++	++	—	B.f.
101	..	+	+	+	++	++	—	B.f.
104	..	+	++	+	++	++	—	B.uf.
109	..	++	++	—	—	—	—	B.uf.
111	..	++	++	++	—	—	—	B.uf.

Case Jackson, A., aged 36.—Patient, who had not been abroad before, left England in July, 1915, and went to the Peninsula, where he remained five months. He was then transferred to Egypt, where on March 23, 1916, he was found to be a carrier of *E. histolytica* during the routine examination of men in Mustapha Camp. He also had an infection of tetramitus. He gave no history of dysentery. He was kept under observation till April 1, when a course of emetin by the mouth (one grain a day in tinc. opii for 12 days) was commenced. During this course patient vomited after the emetin on several occasions. The *E. histolytica* disappeared, but recurred again three days after the course was completed. Patient was then given emetin injections, one grain a day for 12 days. The *E. histolytica* disappeared after the second dose, but recurred again a fortnight after the course was finished. During treatment patient was not kept in bed and was on chicken diet. The two courses of emetin had no effect on the pulse-rate and the temperature was not altered.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.f.	Tet.c.	Tet.f.	
1	..	++	—	+++	—	B.f.	12	E.m.1. V. 5 min.	—	—	+++	—	B.f.
3	..	++	+	+++	—	B.l.							
4	..	—	—	+	—	B.uf.	13	E.m.1. V. inst.	—	—	+	—	B.f
6	..	—	—	++	++	B.uf.							
7	..	++	—	+++	—	B.f.	14	E.m.1	—	—	++	—	B.f.
9	E.m.1	—	—	—	+++	B.uf.	15	E.m.1	—	—	++	—	B.f.
10	E.m.1	—	—	+++	—	B.uf.	16	E.m.1. V. 1 hr.	—	—	—	—	—
11	E.m.1	—	—	—	—	—							

* Note.—When the patient was examined one year later by Mr. Savage in Egypt, cysts of *E. histolytica* were still present in the stool.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.f.	Tet.c.	Tet.f.	
17	E.m.1	++	—	++	+	B.sf.	31	E.1	—	—	+++	—	B.uf.
18	E.m.1	+	—	+++	—	B.uf.	32	E.1	—	—	+++	—	B.uf.
19	E.m.1.	—	—	—	—	—	33	E.1	—	—	+	—	B.f.
	V. 1 hr.						34	E.1	—	—	—	—	—
20	E.m.1	—	—	—	—	B.uf.	35	E.1	—	—	+++	+++	B.uf.
21	..	—	—	—	—	B.uf.	36	E.1	—	—	++	++	B.l.
22	..	—	—	—	—	B.uf.	37	E.1	—	—	—	+++	B.uf.
23	..	++	—	+++	—	B.f.	38 C.	..	—	—	—	+++	B.uf.
25	..	+	+	+++	—	B.uf.	42	..	—	—	—	++	B.f.
26	E.1	+	—	+++	—	B.uf.	45	..	—	—	+++	—	B.f.
27	E.1	—	—	—	—	—	49	..	—	—	+	—	B.f.
28	E.1	+	++	++	+	B.l.	52	..	+	—	+	—	B.f.
29	E.1	—	—	+++	+++	B.uf.	58	..	+	—	+++	—	B.f.
30	E.1	—	—	+++	—	B.uf	63	..	+	+	+++	—	B.f.

Case Main, aged 42.—Patient, who had not been abroad before, left England on May 24, 1915, for the Peninsula, where he remained for six months (Cape Hellas). While there he suffered from diarrhœa, but had no dysentery. He came to Alexandria and was found on March 20, 1916, to be a carrier of *E. histolytica* in the routine examination of men in Mustapha Camp. He was under observation till March 28, when a course of emetin by the mouth (one grain a day for 12 days) was commenced. The *E. histolytica* did not disappear, so the patient was given a course of emetin injections (one grain a day for 12 days) from April 14. The *E. histolytica* cysts quickly disappeared, but recurred 10 days later. The emetin courses had no effect on the temperature or pulse-rate. Trichomonas was also present, and an *E. coli* infection appeared a few days after the patient went to the convalescent camp. During the treatment the patient was not kept in bed and was given chicken diet. The emetin by the mouth caused him to vomit on two occasions only.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.c.c.	E.f.	Trich.				E.h.c.	E.c.c.	E.f.	Trich.	
1	..	++	—	—	—	B.f.	23	..	++	—	+	+++	B.uf.
3	..	—	—	—	+	B.uf.	24	..	++	—	—	+++	B.sf.
4	..	—	—	—	+++	B.uf.	25	E.1	—	—	—	—	—
5	..	—	—	—	+++	B.l.	26	E.1	++	—	—	—	B.sf.
6	..	—	—	+	+++	B.uf.	27	E.1	—	—	—	—	B.l.
7	..	+	—	—	+++	B.f.	28	E.1	—	—	—	—	B.uf.
8	E.m.1. V.¾hr.	++	—	—	++	B.sf.	29	E.1	—	—	—	—	—
9	E.m.1	—	—	—	+++	B.uf.	30	E.1	—	—	—	++	B.uf.
10	E.m.1	—	—	—	—	—	31	E.1	—	—	—	—	B.l.
11	E.m.1	—	—	—	++	B.uf.	32	E.1	—	—	—	—	B.uf.
12	E.m.1	—	—	—	++	B.uf.	33	E.1	—	—	—	—	B.l.
13	E.m.1	—	—	—	++	B.uf.	34	E.1	—	—	—	—	B.uf.
14	E.m.1. V.1hr.	+++	—	++	—	B.uf.	35	E.1	—	—	—	—	B.uf.
15	E.m.1	++	—	+	—	B.uf.	36	E.1	—	—	—	+++	B.uf.
16	E.m.1	—	—	+	+	B.uf.	37 C.	..	—	—	—	++	B.uf.
17	E.m.1	—	—	—	—	—	42	..	—	+	—	—	B.uf.
18	E.m.1	+	—	—	—	B.uf.	44	..	+++	—	—	—	B.f.
19	E.m.1	—	—	—	—	B.uf.	46	..	+++	—	—	—	B.f.
20	..	++	—	++	++	B.uf.	48	..	++	—	—	—	B.f.
21	..	+	—	—	—	B.uf.	50	..	++	—	+	—	B.f.
22	..	+++	—	+	+++	B.l.							

Case Spiers, D., aged (?).—Patient, who had lived in Ceylon for some years, first suffered from dysentery in 1911. He was ill for 10 weeks and the disease was said to be of bacillary

origin. During 1914 and 1915 he had many attacks of amœbic dysentery, for which he was treated as follows:—

1914.	May	1 week ..	12 injections of emetin (6 grains)		
	July	3 days ..	3 ,, ,,	(1½ ,,)	
	August	1 week ..	10 ,, ,,	(5 ,,)	
	October..	1 ,, ..	6 ,, ,,	(3 ,,)	
	December	1 ,, ..	8 ,, ,,	(4 ,,)	
1915.	August	2 days ..	6 ,, ,,	(3 ,,)	
	October..	2 ,, ..	6 ,, ,,	(3 ,,)	
	November	1 week ..	10 ,, ,,	(5 ,,)	
	December	1 ,, ..	10 ,, ,,	(5 ,,)	

During January and February, 1916, patient on two occasions had two injections of emetin (½ grain at each injection) as he felt as if an attack of dysentery was impending. He has also been in the habit of taking a ½-grain keratin-coated tabloid of emetin when any trace of blood and mucus appeared in the stool. This he had done on four occasions. He was convinced that this procedure warded off attacks of dysentery. When seen on February 19, 1916, patient was thin and sallow looking, and was evidently not well. The stool brought for inspection was soft and semi-formed with flakes of mucus distributed through it. There were present enormous numbers of amœbæ, which the subsequent observations proved to be minuta forms of *E. histolytica*. As patient could not very well come into hospital he was treated while on duty (office work) with emetin injections (one grain a day for 12 days). He was put on light chicken diet and was to avoid all alcohol. A stool examined three days after treatment showed *E. histolytica* cysts in enormous numbers. These he continued to pass through the whole course of emetin injections. Emetin by the mouth was then given—one grain at night for two nights and then ½ grain at night for seven nights. After the first dose by the mouth the cysts showed signs of degeneration, while none were found after the second dose. The patient was kept under observation for over a month after treatment. There was no recurrence of the amœbæ in the stool and the patient continued to improve in health. The intestinal symptoms completely cleared up, and patient passed normal stools for the first time since his dysenteric attacks commenced.

Days	Treatment	Findings			Days	Treatment	Findings		
		E.h.c.	E.f.	Stool			E.h.c.	E.f.	Stool
1	..	—	+++	B.sf.	16	E.m.½	—	—	B.uf.
2	E.1	—	+++	B.uf. B.m.	17	E.m.½	—	—	B.uf.
3	E.1	—	—	—	18	E.m.½	—	—	L.b.uf.
4	E.1	—	—	—	19	E.m.½	—	—	Y.l.
5	E.1	+++	—	B.l.	20	E.m.½	—	—	Y.l.
6	E.1	+++	+++	B.uf.	21	E.m.½	—	—	B.l.
7	E.1	+++	+++	B.uf.	22	E.m.½	—	—	B.l.
8	E.1	+++	+++	B.uf.	23	..	—	—	B.l.
9	E.1	—	++	B.uf.	25	..	—	—	B.sf.
10	E.1	++	+	B.l.m.	27	..	—	—	B.uf.
11	E.1	++	—	B.uf.	35	..	—	—	B.sf.
12	E.1	++	—	B.uf.	41	..	—	—	B.uf.
13	E.1	++	++	B.uf.	46	..	—	—	B.uf.
14	E.m.1. V. 2 hrs.	--	+	B.l.	54	..	—	—	B.sf.
15	E.m.1. V. 3 hrs.	—	—	Y.l.					

Case Healy, E., aged 26.—Patient, who had lived in the Federated Malay States, had his first attack of dysentery there in the spring of 1911. He was in hospital three weeks and did not have another attack till 1912, for which he was given emetin. Another attack in 1913 or beginning of 1914 occurred for which emetin was again administered. Patient came home

to England in November, 1914, and was then very fit except for the fact that he always had loose motions. About July, 1915, when under canvas in wet weather in England, he experienced another attack of dysentery for which he went into hospital and was given emetin. He came out of hospital but very soon relapsed again. This time he was treated with rectal injections of silver salts. In November, 1915, he left England for the Mediterranean and had an attack on the ship. He came to Egypt and remained fairly well to the end of the year. When on the way to Mudros he was very ill with dysentery. While in Mudros he was constantly ill, and after a fortnight was sent back to Egypt. Up to February 23, 1916, when the stool was first examined microscopically, patient was constantly passing blood and mucus. When examined on this date the stool was found to consist of brown soft fæcal matter with some patches of blood and mucus, and contained numerous small amœbæ which looked like minuta forms of *E. histolytica*. A similar condition was observed next day, while on the next day after this, in addition to the small amœbæ, there were numerous cysts of *E. histolytica*. Patient was placed on light diet and given a course of emetin injections, one grain a day from February 25, to March 7. This had practically no effect on the infection. Owing to the good result obtained in another case (Spiers) patient was then given emetin by the mouth. From February 9 he had ½ grain a day for four days and then one grain a day for six days. As this treatment also gave no result patient was then given a course of pulv. ipecac., commencing with 30 grains. This was decreased daily by five grains till a dose of 10 was reached, when patient continued the 10 grains a day for eight days. As this treatment was in no way reducing the infection, patient was given an injection of emetin one ½ grain a day along with the 10 grains of pulv. ipecac. As no change in the infection was produced, this double treatment was discontinued after three days. Owing to a suggestion that thymol was toxic to amœbæ, this drug in doses of 10 grains t.d.s. was tried for eight days. This drug yielded no better results than the others tried before. During the whole of this time patient was not kept in bed and was mostly on chicken diet. It was accordingly decided to try emetin treatment by the mouth in larger doses, to keep the patient in bed, and to give him a rigid dysentery diet of arrowroot water, jelly, tea with milk, and a little custard. This diet was commenced two days before the emetin was started. Patient then took 1½ grains of emetin each night for 12 nights. After three days of this treatment the amœbæ disappeared from the stool and patient was allowed towards the end of the course a little extra food in the shape of beef tea, an egg and rusks. The emetin was kept down without vomiting, except on the first occasion, though this necessitated a heroic struggle on the patient's part. Three days after the end of this course of emetin there were as many cysts of *E. histolytica* in the stool as before treatment. These, with or without minuta forms of *E. histolytica*, were found regularly till May 13, when patient was invalided home to England. The large quantity of emetin taken by the patient had very little effect on his general health, and any weakness after treatment could be attributed to the restricted diet and stay in bed during the last course of emetin. The patient had a pure infection of *E. histolytica* uncomplicated by the presence of any other protozoa.

Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool
		E.h.c.	E.f.				E.h.c.	E.f.	
1	..	—	+++	B.uf.B.m.	10	E.1	—	—	—
2	..	—	+++	B.uf.	11	E.1	+	+++	B.uf.
3	E.1	+++	+++	B.uf.	12	E.1	+++	++	B.uf.
4	E.1	—	—	—	13	E.1	—	—	—
5	E.1	+++	—	B.l.	14	E.1	++	—	B.uf.
6	E.1	+++	+++	B.l.	15	..	+	++	B.uf.
7	E.1	+++	+++	B.l.	16	E.m.½	+	++	B.uf.
8	E.1	—	+++	B.l.	17	E.m.½	+	++	B.uf.
9	E.1	+	++	B.uf.	18	E.m.½	+++	++	B.uf.

Days	Treatment	Findings E.h.c.	Findings E.f.	Stool	Days	Treatment	Findings E.h.c.	Findings E.f.	Stool
19	E.m.½	+++	—	B.uf.	44	Thy. gr. 10	+++	—	B.uf.
20	E.m.1	+++	++	B.uf.	45	Thy. gr. 10	++	+	B.uf.
21	E.m.1	—	—	—	46	Thy. gr. 10	+++	++	B.uf.
22	E.m.1	+++	—	B.uf.	47	..	+++	—	B.uf.
23	E.m.1	—	—	—	48	..	+++	—	B.uf.
24	E.m.1	—	—	—	49	E.m.1½ V. 6 hrs.	+++	++	B.l.
25	E.m.1	+++	+++	B.l.	50	E.m.1½	+++	+	B.l.
26	P. ipecac. gr. 30	+++	+++	B.l.	51	E.m.1½	+	—	B.l.
27	P. ipecac. gr. 25	—	—	—	52	E.m.1½	+	—	B.l.
28	P. ipecac. gr. 20	++	++	L.b.l.	53	E.m.1½	—	—	B.uf.
29	P. ipecac. gr. 15	++	++	L.b.l.	54	E.m.1½	—	—	B.uf.
30	P. ipecac. gr. 10	+	+	L.b.l.	55	E.m.1½	—	—	B.l.
31	P. ipecac. gr. 10	—	—	—	56	E.m.1½	—	—	L.b.l.
32	P. ipecac. gr. 10	—	—	B.l.	57	E.m.1½	—	—	L.b.l.
33	P. ipecac. gr. 10	+++	+++	L.b.l.	58	E.m.1½	—	—	L.b.uf.
34	P. ipecac. gr. 10	—	+++	L.b.l.	59	E.m.1½	—	—	L.b.uf.
35	P. ipecac. gr. 10 E.½	—	+++	L.b.l.	60	E.m.1½	—	—	L.b.uf.
36	P. ipecac. gr. 10 E.½	++	—	L.b.l.	61	..	—	—	L.b.uf.
37	P. ipecac. gr. 10 E.½	—	—	—	62	..	—	—	B.uf.
38	E.½	++	+++	L.b.uf.	63	..	++	—	B.uf.
39	Thy. gr. 10	+++	+++	B.uf.	64	..	+++	—	B.uf.
40	Thy. gr. 10	+++	++	B.uf.	68	..	+++	—	B.uf.
41	Thy. gr. 10	+++	++	B.uf.	72	..	+++	—	B.uf.
42	Thy. gr. 10	+++	++	B.uf.	76	..	+++	++	B.uf.
43	Thy. gr. 10	++	+++	B.uf.	78	..	++	+++	B.uf.

Note.—On his arrival in England, the patient was seen by Dr. A. C. Stevenson and Dr. G. C. Low, who undertook the further treatment. A course of bismuth emetin iodide (3 grains a day for 12 days) was given, with the result that the *E. histolytica* infection entirely disappeared, and did not recur during a control of 10½ months with 38 examinations. A tetramitus infection which had developed did not yield to the treatment.

Case Kettlewell, aged 24.—Patient, who had not been abroad before, left England in June, 1915, and went direct to Gallipoli. He was there for seven weeks but had dysentery after having been there two weeks. He remained on duty, however, till he was compelled to report sick in September. He was invalided to Malta, where he remained one month and suffered from jaundice. Thence he returned to England and was in hospital a few days till he was discharged. He was given six weeks leave, but was ill part of the time with dysenteric symptoms. He was back on duty (A class) in December, 1915, and returned to the Mediterranean Egypt in February. Since the beginning of his illness patient had had diarrhœa or dysentery continuously till December, when there was some improvement. He was never treated with emetin. On March 1 and 3, owing to continued looseness of the bowels, patient's stool was examined and he was found to have a large *E. histolytica* infection, together with tetramitus and *E. coli*. Patient was taken into hospital and given a course of emetin injections of one grain a day for 12 days. The *E. histolytica* infection was not abolished by this treatment, during which patient was not kept in bed and was on chicken diet. It was accordingly decided to keep patient in bed, give him daily a rigid dysentery diet (tea with milk, arrowroot, jelly, and a little custard), and institute a course of emetin by the mouth. Patient was given in this way emetin in tinc. opii 1½ grains a day for four days, followed by one grain a day for eight days. There was no vomiting. The *E. histolytica* quickly disappeared, as also did the tetramitus and *E. coli*. There was no return of the *E. histolytica* infection during a control of one month after treatment. The other infections, however, recurred. When patient got up after the second course of emetin he felt very weak and suffered from marked shortness of breath and irregular action of the heart on slight exertion. This condition continued for about one month and gradually disappeared, the patient finally returning to duty. In this case it appeared as if the emetin administered had had some bad effect upon the heart.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	
1	..	++	+	++	—	++	B.l.	23	E.m.1½	+	++	++	+	—	B.uf.
3	..	+++	—	+++	—	—	B.uf.	24	E.m.1½	—	—	+	—	+	B.uf.
4	..	—	—	+++	—	+	B.uf.	25	E.m.1	—	+	—	—	++	B.uf.
5	E.1	+++	+	—	—	—	B.f.	26	E.m.1	—	—	—	—	++	B.uf.
6	E.1	—	—	—	—	—	—	27	E.m.1	—	—	—	—	—	B.uf.
7	E.1	—	+	++	+	+++	B.sf.	28	E.m.1	—	—	—	—	—	B.uf.
8	E.1	—	—	—	—	—	—	29	E.m.1	—	—	—	—	—	B.l.
9	E.1	—	++	++	—	+++	B.uf.	30	E.m.1	—	—	—	—	—	L.b.l.
10	E.1	—	—	—	—	—	—	31	E.m.1	—	—	—	—	—	L.b.l.
11	E.1	+	++	+	—	++	B.uf.	32	E.m.1	—	—	—	—	—	—
12	E.1	++	++	+++	++	+++	B.uf.	33	..	—	—	—	—	—	L.b.l.
13	E.1	+	+	+++	—	++	B.uf.	34	..	—	—	—	—	—	B.uf.
14	E.1	+	+	—	+	—	B.uf.	40	..	—	—	—	—	—	B.f.
15	E.1	+	++	+++	—	+++	B.uf.	43	..	—	+	+	—	—	B.f.
16	E.1	—	+	+++	—	++	B.uf.	47	..	—	—	—	—	—	B.uf.
18	..	+++	++	+++	—	+++	B.uf.	51	..	—	++	—	++	—	B.uf.
19	..	++	++	++	+++	++	B.uf.	54	..	—	+	—	—	++	B.uf.
20	..	+	++	+++	++	++	B.uf.	59	..	—	—	—	—	—	B.uf.
21	E.m.1½	—	+++	—	+++	—	B.f.	61	..	—	—	—	—	—	B.uf.
22	E.m.1½	—	—	—	—	—	—	64	..	—	++	—	—	—	B.f.

Case Bennett, J., aged 47.—Patient, who had not been abroad before, left England in 1915 for the Peninsula, where he remained from May to October. He was transferred to Egypt, and on April 16, 1916, during the routine examination of cooks in Mustapha Camp was found to be a carrier of *E. histolytica*. There was also an infection of *E. coli*, and later a trichomonas infection appeared. There was no history of dysentery. Patient was kept under observation till April 25, when a course of emetin injection was commenced, one grain a day for 12 days. The *E. histolytica* cysts did not disappear from the stool, so from May 9 onwards for 12 days patient was given daily by the mouth a grain of emetin in keratin-coated tabloid. There was no vomiting, and the *E. histolytica* disappeared after the second day of treatment. There was no recurrence of the infection during a control of one month, three weeks of which patient spent in the convalescent camp, where he performed light duty. While there a trichomonas infection manifested itself. During the course of emetin patient was not kept in bed and was on chicken diet. The emetin had no effect on patient's temperature or pulse-rate.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.c.c.	E.f.	Trich.				E.h.c.	E.c.c.	E.f.	Trich.	
1	..	+	+	—	—	B.f.	28	E.m.1	—	—	—	—	L.b.uf.
3	..	+	—	—	—	B.f.	29	E.m.1	—	—	—	—	B.uf.
5	..	+	—	—	—	B.uf.	30	E.m.1	—	—	—	—	L.b.uf.
6	..	—	—	—	—	B.uf.	31	E.m.1	—	—	—	—	B.l.
7	..	—	—	—	—	B.uf.	32	E.m 1	—	—	—	—	L.b.f.
8	..	—	—	++	—	B.uf.	33	E.m.1	—	—	—	—	B.uf.
9	..	—	—	—	—	B.uf.	34	E.m.1	—	—	—	—	B.uf.
10	E.1	+	+	—	—	B.uf.	35	E.m.1	—	—	—	—	B.uf.
11	E.1	+++	+++	—	—	B.uf.	37	..	—	—	—	—	B.uf.
12	E.1	++	++	—	—	B.uf.	39	..	—	—	—	+	B.uf.
13	E.1	++	+	—	—	B.uf.	40	..	—	—	—	+++	B.uf.
14	E.1	++	+	—	—	B.uf.	41 C.	..	—	—	—	—	—
15	E.1	++	+	—	—	B.uf.	42	..	—	—	—	—	B.f.
16	E.1	—	++	+	—	B.sf.	44	..	—	—	—	+++	B.uf.
17	E.1	—	+	—	—	B.uf.	46	..	—	—	—	+	B.uf.
18	E.1	—	++	—	—	B.uf.	48	..	—	+	—	—	B.uf.
19	E.1	++	++	—	—	B.uf.	52	..	—	—	—	—	B.sf.
20	E.1	++	++	—	—	B.uf.	54	..	—	—	—	++	B.uf.
21	E.1	+	++	—	—	B.uf.	56	..	—	—	—	—	B.sf.
22	..	+	++	—	—	B.uf.	58	..	—	—	—	—	B.sf.
23	..	++	—	—	—	B.f.	60	..	—	—	—	+	B.sf.
24	E m.1	+	+	—	—	B.sf.	62	..	—	—	—	+	B.sf.
25	E.m.1	++	+	—	—	B.f.	64	..	—	—	—	—	B.sf.
26	E.m.1	—	+	—	—	L.b.uf.	66	..	—	—	—	—	B.sf.
27	E.m.1	—	+	—	—	L.b.uf.							

Case Obbard, A., aged 21.—Patient, who had never been abroad before, left England on June 1, 1915, and went direct to the Peninsula (Cape Hellas and Suvla Bay). While there he had attacks of diarrhœa, was wounded, and finally transferred to Alexandria. On April 2, 1916, during the routine examination of cooks in Mustapha Camp, patient was found to be a carrier of *E. histolytica*. He had also an infection of *E. coli*. There was no history of dysentery and patient had never had emetin. He was kept under observation till April 11, when a course of emetin injections (one grain a day for 12 days) was commenced. The cysts of *E. histolytica* disappeared at first but they recurred before the end of the course and were just as numerous afterwards. Patient was observed till May 3 when a second course of emetin for 12 days was given (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth at night). The day after treatment was commenced no cysts of *E. histolytica* were found and they did not recur during a control of 48 days after the course was completed. Unfortunately no stool was examined on the day the course was commenced, but the *E. histolytica* cyst had been passed regularly up to that date. During the first course of emetin patient was not kept in bed and was given chicken diet, while during the second course he was in bed on milk diet. The emetin by the mouth caused vomiting on three occasions. During the first course of emetin there was a slight increase in the patient's pulse-rate, which persisted after the treatment for a few days. During the second course the increase in pulse-rate was still more marked, and it did not subside for at least a fortnight, and patient complained of shortness of breath on exertion. There was no change in the temperature.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	+	++	—	B.f.	41	E.1 E.m.½	—	—	—	L.b.f.
4	..	+	++	+	B.uf.	42	E.1 E.m.½ V. 1 hr.	—	—	—	B.l.
5	..	++	++	—	B.f.						
6	..	—	+	—	B.f.	43	E.1 E m.½	—	—	—	B.uf.
8	..	—	+	—	B.f.	44	..	—	—	—	B.sf.
9	..	++	++	—	B.f.	45	..	—	—	—	B.uf.
10	E.1	++	+++	—	L.b.uf.	46	..	—	—	—	B.uf.
11	E.1	++	+++	—	B.uf.	47	..	—	—	—	B.uf.
12	E.1	—	++	—	L.b.uf.	49	..	—	—	—	B.uf.
13	E.1	—	+	—	B.f.	51	..	—	+	—	B.f.
14	E.1	—	++	—	B.f.	53	..	—	—	—	B.f.
15	E.1	—	++	—	B.l.	54	..	—	—	—	B.f.
16	E.1	+	++	—	B.uf.	56	..	—	++	++	B.f.
17	E.1	—	++	—	B.f.	57	..	—	+++	+	B.uf.
18	E.1	—	+++	—	B.f.	58	..	—	+++	—	B.f.
19	E.1	—	+	—	B.f.	59	..	—	+	—	B.uf.
20	E.1	—	++	—	B.f.	60	..	—	+	—	B.sf.
21	E.1	+	+	—	B.f.	61	..	—	++	—	B.f.
22	..	+++	+++	—	B.uf.	63	..	—	++	—	B.sf.
23	..	+++	+++	—	B.uf.	65	..	—	—	—	B.uf.
24	..	+++	+++	—	B.uf.	67	..	—	—	—	B.f.
26	..	+++	+++	—	B.uf.	68	..	—	++	++	B.uf.
27	..	+++	+++	—	B.uf.	69	..	—	+	++	B.uf.
28	..	+++	+	—	B.uf.	71	..	—	+	—	B.f.
29	..	—	—	++	B.uf.	74	..	—	++	+	B.uf.
30	..	++	+	+	B.uf.	75	..	—	+	++	B.uf.
31	..	++	+	—	B.uf.	76	..	—	—	++	B.f.
32	E.1 E.m.½	—	—	—	—	77	..	—	—	—	B.f.
33	E.1 E.m.½ V. 1 hr.	—	—	—	B.uf.	80	..	—	++	++	B.f.
						82	..	—	—	—	B.uf.
34	E.1 E.m.½	—	+	—	B.uf.	84	..	—	—	+++	B.uf.
35	E.1 E.m.½	—	—	—	L.b.uf.	86	..	—	—	—	B.uf.
36	E.1 E.m.½	—	—	—	L.b.uf.	87	..	—	—	—	B.f.
37	E.1 E.m.½	—	—	—	L.b.uf.	88	..	—	—	—	B.f.
38	E.1 E.m.½	—	—	—	B.uf.	89	..	—	—	—	B.f.
39	E.1 E.m.½	—	—	—	B.f.	90	..	—	—	+	B.f.
40	E.1 E.m.½ V. 45 min.	—	—	—	L.b.sf.	91	..	—	—	+	B.f.

Case Dorter, P., aged 32.—Patient, who is an Australian, first had dysentery two years ago in Western Australia, and has had attacks on many occasions since then. In March, 1915, he was ill with dysentery in Cairo, and later in the month in hospital in Alexandria. He was treated with emetin (dose ?). During April and May, 1915, he had dysentery on the Peninsula. He was invalided to Malta with a gunshot wound in the hand, and had an attack of dysentery while there. He had further attacks in England in November, 1915, and March, 1916. He was transferred to Egypt and suffered from dysentery on the voyage out. He was admitted to the 15th General Hospital on arrival at Alexandria on April 10, 1916. Between April 10 and April 24 the patient's stool was examined four times with negative results, but on the latter date the stool consisted of much blood and mucus mixed with brown fæcal matter. There were present a few cysts of *E. histolytica* and a fair number of active amœbæ, some of which contained red blood corpuscles. Patient was given a course of emetin injections of one grain a day for 12 days. The infection disappeared after four days' treatment, during which patient was kept in bed and placed on milk diet. Patient went to the convalescent camp five days after treatment was finished, and five days later was again passing cysts of *E. histolytica* and free amœbæ. An infection of *E. coli* had also appeared. Patient was re-admitted to hospital (19th General), and on May 25 a second 12-day course of emetin was commenced (one grain injection each morning and ½ grain by the mouth in keratin-coated tabloid each night). Patient was kept in bed and placed on milk diet during the treatment. The infection again disappeared, and patient was sent to the convalescent camp on June 11, where *E. histolytica* cysts again appeared in the stool 10 days after the course of emetin was completed. The emetin courses had no effect on the temperature or pulse-rate of the patient.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	E.1	+	—	++r.b.c.	B.uf. B.m.	32	E.1 E.m.½	—	—	—	B.uf.
2	E.1	—	—	+++r.b.c.	B.uf. B.m.	33	E.1 E.m.½	—	—	—	B.uf.
3	E.1	—	—	+++	B.uf. B.m.	34	E.1 E.m.½	—	—	—	—
4	E.1	—	—	+	B.uf. B.m.	35	E.1 E.m.½	—	—	—	B.uf.
5	E.1	—	—	+++	B.uf. B.m.	36	E.1 E.m.½	—	—	—	B.uf.
6	E.1	—	—	—	—	37	E.1 E.m.½	—	—	—	B.uf.
7	E.1	—	—	—	B.uf.	38	E.1 E.m.½	—	—	—	B.f.
8	E.1	—	—	—	B.uf. B.m.	39	E.1 E.m.½	—	—	—	B.f.
9	E.1	—	—	—	B.uf.	40	E.1 E.m.½	—	—	—	B.f.
10	E.1	—	—	—	B.uf.	41	E.1 E.m.½	—	—	—	B.sf.
11	E.1	—	—	—	B.uf.	42	E.1 E.m.½	—	—	—	B.uf.
12	E.1	—	—	—	B.uf.	43	..	—	—	—	B.sf.
13	..	—	—	—	B.uf.	44	..	—	—	—	B.uf.
15C.	..	—	—	—	—	45	..	—	—	—	B.uf.
16	..	—	+++	+	B.uf.	46	..	—	—	—	B.uf.
21	..	++	+	+	B.f.	47	..	—	—	—	B.uf.
23	..	++	+	+	B.uf.	48	..	—	—	—	B.sf.
25	..	++	+	++	B.f.	49	..	—	—	—	B.sf.
27	..	++	—	+	B.uf.	51	..	—	—	—	B.sf.
29	..	++	—	—	B.sf.	53	..	++	—	+	B.sf.
31	E.1 E.m.½	++	—	—	B.f.	55	..	++	++	—	B.sf.

Case Ball, aged 36.—Patient, who had not been abroad before, left England in 1915 for Mudros, where he remained nine months. He was transferred to Egypt, and on March 22, 1916, while acting as head cook at Orwa-el-Waska Hospital, he was taken ill with dysentery. He had had three previous attacks of dysentery for which he had been in hospital. In Mudros he had one injection of emetin (dose ?). When admitted to hospital on March 22, the stool consisted of fæcal matter and a large quantity of dark brown blood-stained mucus. The mucus showed numerous large active amœbæ, many of which contained red blood corpuscles.

The fæcal portion contained numerous smaller amœbæ (minuta forms of *E. histolytica*) and cysts of *E. histolytica*, together with tetramitus and trichomonas. The case was undoubtedly one of a carrier relapsing into an attack of acute dysentery. On the following day a similar stool was passed, and patient was put on a course of emetin by the mouth (one grain a day for 12 days). The dysentery quickly cleared up and the amœbæ disappeared after the second dose, but not the trichomonas and tetramitus. Patient vomited only on one occasion—30 minutes after the second dose of emetin. During treatment patient was on liquid diet and was kept in bed. After treatment he returned to duty in the hospital kitchen, where he was kept under observation. Eleven days after the course of emetin was completed, *E. histolytica* (free and encysted) were found in the stool. Patient was then given a course of emetin injections one grain a day for 12 days. The *E. histolytica* again disappeared but not the tetramitus or trichomonas. During this course patient was on full diet and continued duty as cook. Twenty-four days after this course was finished *E. histolytica* again appeared in the stool and persisted to the end of the observation, when patient was sent home to England. During the latter part of the observation patient had an infection of the small flagellate hitherto undescribed *Tricercomonas intestinalis*.

Days	Treatment	Findings						Stool
		E.h.c.	E.f.	Tet.c.	Tet.f.	Tr'ch.	T.c.	
1	..	+	+++ r.b.c.	—	+	++	—	B.uf. B.m.
2	E.m.1	+++	+++ r.b.c.	—.	—	+	—	B.uf. B.m.
3	E.m.1. V. 30 min.	—	+	—	—	—	—	L.b.l.
4	E.m.1	—	—	—	—	—	—	B.uf.
5	E.m.1	—	—	—	—	—	—	B.f.
6	E.m.1	—	—	—	++	++	—	B.f.
7	E.m.1	—	—	—	+	+	—	B.l.
8	E.m.1	—	+	+++	+++	+++	—	L.b.uf.
9	E.m.1	—	—	+	++	++	—	B.uf.
10	E.m.1	—	—	+	++	—	—	B.uf.
11	E.m.1	—	—	—	++	++	—	B.uf.
12	E.m.1	—	—	—	++	++	—	B.uf.
13	E.m.1	—	—	—	—	—	—	—
14	..	—	—	—	++	++	—	B.l.
19	..	—	—	—	—	++	—	B.uf.
24	..	++	++	—	—	—	—	B.uf.
25	..	+++	—	—	++	++	—	B.uf.
30	E.1	—	—	—	—	—	—	—
31	E.1	—	+	+	+	+++	—	B.uf.
32	E.1	—	—	—	+++	+++	—	B.uf.
33	E.1	—	—	—	++	++	—	B.uf.
34	E.1	—	—	—	—	—	—	—
35	E.1	—	+	—	—	—	—	B.uf.
36	E.1	—	—	—	+	+	—	B.uf
37	E.1	—	—	—	++	+	—	B.uf.
38	E.1	—	—	—	+++	++	—	B.uf.
39	E.1	—	—	—	—	—	—	—
40	E.1	—	—	—	+++	++	—	B.uf.
41	E.1	—	—	—	+++	++	—	B.uf.
43	..	—	—	—	+++	++	—	B.uf.
46	..	—	—	—	—	—	—	B.uf.
48	..	—	—	—	—	+++	—	B.f.
49	..	—	—	—	—	—	—	B.f.
50	..	—	—	—	—	++	—	B.f.
52	..	—	—	—	—	—	—	B.f.
54	..	—	—	—	+++	++	—	B.f.
56	..	—	—	—	+++	+++	—	L.b.uf.
58	..	—	—	—	—	++	—	B.f.
60	..	—	—	—	—	—	—	B.f.
61	..	—	—	—	—	—	—	B.f.
62	..	—	—	—	+++	++	—	B.f.

Days	Treatment	Findings						Stool
		E.h.c.	E.f.	Tet.c.	Tet.f.	Trich.	T.c.	
63	..	—	—	—	—	++	—	B.sf.
64	..	—	—	—	+++	+++	—	B.uf.
65	..	+	++	++	+++	+++	—	B.uf.
66	..	+	++	+++	++	+++	—	B.uf.
68	..	+	+	—	+++	+++	—	B.uf.
69	..	++	—	+++	++	+++	+++	B.uf.
70	..	++	—	+++	++	+++	+++	B.uf.
71	..	++	—	+++	+++	+++	++	B.uf.
72	..	++	—	++	+++	+++	—	B.uf.
73	..	++	++	++	+++	+++	—	B.uf.
74	..	+	+	—	+++	++	—	B.uf.
76	..	++	+	—	+++	++	—	B.uf.

Case Barrie, D. W., aged 43.—Patient left Australia in December, 1914, and was eight months on the Peninsula where he had dysentery, for which he received eight injections of emetin (dose ?). In February, 1916, patient, who had come to Egypt, was in hospital for dysentery. On April 4 he was again admitted for dysentery, and was passing stool consisting of blood and mucus with active amœbæ. On the same day a course of emetin by the mouth was commenced (one grain a day for 12 days). On the fifth day of treatment cysts of *E. histolytica* were found; these persisted all through the treatment by the mouth, and when 12 doses had been given a course of emetin injections was commenced (one grain a day for 12 days). The *E. histolytica* infection disappeared two days before the end of this course, but they appeared again within a week. During the second course of emetin a tetramitus infection manifested itself. The two courses of emetin had no effect on the temperature or pulse-rate. During the first course of emetin the patient was in bed on milk diet. During the second course he was not kept in bed and was on chicken diet as actual dysenteric symptoms had disappeared. An *E. coli* infection appeared five days after patient went to the convalescent camp.

Days	Treatment	Findings					Stool	Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.				E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	
1	E.m.1	—	—	+	—	—	B.m.	15	E.1	—	—	++	—	—	B.uf.
2	E.m.1	—	—	++	—	—	B.uf.	16	E.1	—	—	++	++	+++	B.uf.
3	E.m.1	—	—	+	—	—	B.m.	17	E.1	—	—	—	+	+++	B.uf.
4	E.m.1	—	—	—	—	—	L.b.l.	18	E.1	—	—	—	+	+++	B.uf.
5	E.m.1	—	—	—	—	—	—	19	E.1	—	—	—	—	+++	B.uf.
6	E.m.1	—	—	—	—	—	B.sf.	20	E.1	—	—	—	—	++	B.uf.
7	E.m.1	++	—	—	—	—	B.uf.	21	E.1	+++	—	—	++	+++	B.uf.
8	E.m.1	++	—	—	—	—	B.uf.	22	E.1	—	—	+	—	+++	B.uf.
9	E.m.1	+	—	—	—	—	B.sf.	23	E.1	—	—	—	—	++	B.uf.
10	E.m.1	++	—	—	—	—	B.uf.	24	E.1	—	—	—	—	++	B.uf.
11	E.m.1	+	—	—	—	—	B.uf.	25 C.	..	—	—	—	—	—	—
12	E.m.1	++	—	—	—	—	B.uf.	27	..	—	—	—	—	++	B.sf.
13	E.1	+++	—	—	—	—	B.uf.	29	..	++	+	—	—	—	B.uf.
14	E.1	+++	—	—	—	—	B.uf.	31	..	+	+	—	—	—	B.uf.

Case Smith, C., aged 27.—Patient, who had previously served in South Africa and China, left England in March, 1914, for Egypt, where he remained till April, 1915, when he went to the Peninsula. He was there for seven months, and had his first attack of dysentery in November, for which he was invalided to Egypt. On the ship he was given emetin for eight days (two injections a day: dose ?). He was admitted into hospital in Cairo, and given a further course of emetin for seven days (one injection a day: dose ?). Still with some dysenteric symptoms, patient was sent to a convalescent camp, where he was given a third course of emetin

(one injection a day for eight days). The dysentery cleared up, and he went to Mustapha Camp, where, on April 3, 1916, another attack of dysentery developed, and he was admitted to the 15th General Hospital. The stool contained much blood and mucus, and there were present many active free amœbæ, none of which contained red blood corpuscles. Two days later, however, many amœbæ had included red blood cells. Patient was given a 12-day course of emetin injection (one grain a day). The amœbæ disappeared after the first injection, and the dysenteric symptoms subsided, the stool becoming free from blood and mucus. After treatment patient was kept under observation in hospital for three weeks, during which time a stool with blood and mucus and active amœbæ were found on one occasion. Free amœbæ and cysts of *E. coli* and tetramitus were found from time to time. The patient went to convalescent camp on May 12, and on May 19 had another attack of dysentery, for which he was admitted to the Orwa-el-Waska Hospital. The stool contained many active amœbæ with included red blood corpuscles and tetramitus. Patient was then given a 12-day course of emetin (one grain injection each morning and ½ grain in keratin-coated tabloid each evening). Under this treatment the dysentery again subsided, but a week later active amœbæ with included red blood corpuscles were again present in large numbers. Methyl emetin sulphate was then given for 12 days (one grain injection each morning, and one grain in keratin-coated tabloid by the mouth each evening). The patient's symptoms abated, but amœbæ, which, on account of included red blood cells, were certainly *E. histolytica*, did not disappear. During the courses of emetin, the patient was kept in bed, and was given either milk or some stringent dysentery diet. Neither the emetin nor the methyl emetin by the mouth caused any vomiting.

The patient's temperature and pulse-rate were not altered by the treatment, though there was some fever on one occasion, associated with a kind of bilious attack. This case, which has been treated with emetin from the commencement of the disease, has proved very resistant. An interesting feature of the case is the constant absence of cysts of *E. histolytica*, though most careful search has been made for them on many occasions. The only cysts seen have been those of *E. coli*, and one might argue from this that all the amœbæ belonged to this species. The irregularity of the tetramitus infection is also worthy of note.*

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	
1	..	—	—	+++	—	—	B.m.
2	..	—	—	++	—	—	B.l.B.m.
3	E.1	—	—	+++ r.b.c.	—	—	B.l.B.m.
4	E.1	—	—	—	—	—	B.uf.
5	E.1	—	—	—	—	—	B.uf.
6	E.1	—	—	—	—	—	B.uf.
7	E.1	—	—	—	—	—	B.uf.
8	E.1	—	—	—	—	—	B.uf.
9	E.1	—	—	—	—	—	B.uf.
10	E.1	—	—	—	—	—	B.uf.
11	E.1	—	—	—	—	—	B.uf.
12	E.1	—	—	—	—	—	B.uf.
13	E.1	—	—	—	—	—	B.uf.
14	E.1	—	—	—	—	—	B.uf.
15	..	—	—	—	—	—	B.uf.
16	..	—	—	—	—	—	B.uf.
17	..	—	—	—	—	—	B.uf.
18	..	—	+	+	—	+	B.uf.
19	..	—	—	—	—	—	B.uf.
20	..	—	—	—	—	—	B.uf.
21	..	—	—	+	—	—	B.uf.B.m.

* Note.—The patient, who was invalided to England, suffered from dysentery on the journey home. He was treated with emetin. At the dysentery depot at Barton a full course of bismuth emetin iodide (3 grains a day for 12 days) was followed by relapse. A second course of this drug gave no better result. A liver abscess developed, and the patient died after operation. During this period free forms of *E. histolytica* alone were found. Cysts were never seen. Post-mortem findings were, unfortunately, not available.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	
22	..	—	—	+	—	+	B.l.
23	..	—	—	—	—	—	B.uf.
24	..	—	+	+	—	—	B.l.
26	..	—	—	—	—	—	B.uf.
27	..	—	—	—	—	—	B.uf.
28	..	—	—	—	—	—	B.uf.
29	..	—	+	+	—	—	B.uf.
30	..	—	+	+	—	—	B.uf.
31	..	—	—	—	—	—	B.f.
32	..	—	—	—	—	—	B.f.
35	..	—	—	—	—	—	B.uf.
37	..	—	—	—	—	—	L.b.sf.
41	..	—	—	—	—	—	B.uf.
44	..	—	—	+++ r.b.c.	—	+	B.uf.B.m.
47	..	—	—	+++ r.b.c.	—	+	B.uf.
48	..	—	—	++	—	—	B.l.
49	E.1 E.m.½	—	—	+++ r.b.c.	—	++	L.b.uf.B.m.
50	E.I E.m.½	—	—	++ r.b.c.	—	—	B.l.
51	E.1 E.m.½	—	—	++ r.b.c.	—	—	L.b.l.
52	E.1 E.m.½	—	—	—	—	—	L.b.l.
53	E.1 E.m.½	—	+	—	—	—	B.uf.
54	E.1 E.m.½	—	++	++	+	—	B.uf.
55	E.1 E.m.½	—	—	—	—	—	B.uf.
56	E.1 E.m ½	—	—	+	—	—	B.l.
57	E.1 E.m.½	—	—	+	—	—	B.l.
58	E.1 E.m.½	—	—	—	—	—	B.l.
59	E.1 E.m.½	—	—	—	—	—	B.uf.
60	E.1 E.m.½	—	—	+	—	—	B.uf.
61	..	—	—	—	—	—	B.uf.
62	..	—	—	+	—	—	B.uf.
63	..	—	—	—	—	—	B.uf.
64	..	—	—	++	—	—	B.uf.
65	..	—	—	—	—	—	B.l.
66	..	—	—	+++ r.b.c.	—	—	B.uf.
67	..	—	—	++	—	—	B.uf.
68	M.E.1 M.E.m.1	—	—	++	—	+	B.sf.
69	M.E.1 M.E.m.1	—	—	++ r.b.c.	—	—	B.uf.m.
70	M.E.1 M.E.m.1	—	—	+++ r.b.c.	—	—	B.uf.
71	M.E.1 M.E.m.1	—	—	+	—	—	B.uf.
72	M.E.1 M.E.m.1	—	—	+ r.b.c.	—	—	B.uf.
73	M.E.1 M.E.m.1	—	—	—	—	—	B.uf.
74	M.E.1 M.E.m.1	—	—	++	—	—	B.uf.
75	M.E.1 M.E.m.1	—	—	++	—	—	B.uf.
76	M.E.1 M.E.m.1	—	—	++	—	—	B.uf.
77	M.E.1 M.E.m.1	—	—	—	—	—	B.uf.
78	M.E.1 M.E.m.1	—	—	++	—	—	B.uf.
79	M.E.1 M.E.m.1	—	—	—	—	—	B.uf.
80	..	—	—	+++ r.b.c.	—	++	B.uf.B.m.
81	..	—	—	—	—	—	B.uf.
82	..	—	—	+	—	—	B.uf.
83	..	—	—	—	—	—	B.uf.
84	..	—	—	—	—	—	B.uf.
85	..	—	—	+	—	—	B.uf.
86	..	—	—	++	—	—	B.uf.
87	..	—	—	+	—	—	B.uf.
88	..	—	—	++ r.b.c.	—	+	B.uf.m.
89	..	—	—	++	—	—	B.uf.m.
90	..	—	—	+	—	—	B.uf.m.
91	..	—	—	++	—	—	B.uf.m.
92	..	—	—	+++	—	—	B.uf.m.
93	..	—	—	—	—	—	B.uf.
94	..	—	—	—	—	—	B.uf.
95	..	—	—	—	—	—	B.uf.
96	..	—	—	+++	—	++	B.uf.m.
97	..	—	—	+++	—	+	B.uf.m.
98	..	—	—	+++	—	—	B.uf.
99	..	—	—	+++ r.b.c.	—	++	B.uf.B.m.
100	..	—	—	+++ r.b.c.	—	++	B.uf.B.m.
101	..	—	—	+++ r.b.c.	—	+	B.uf.B.m.

Case Rushforth, aged 33.—Patient, who had not been abroad before, left England on December 5, 1915, and came direct to Egypt, where he remained. On April 24, 1916, he was admitted to the 15th General Hospital with a history of diarrhœa for nine days and the passage of blood and mucus for two days. There was no previous history of dysentery. The case was evidently one of a primary attack of amœbic dysentery. The stool contained not only active free amœbæ but also cysts of *E. histolytica*. It would appear from the presence of the cysts that the patient had become a carrier and had then lapsed into the condition of acute dysentery. Patient was given a course of emetin injections of one grain a day for 12 days. He was kept in bed on dysentery diet. The *E. histolytica* quickly disappeared, but during the course an *E. coli* infection became evident, and within a fortnight of the completion of the course the *E. histolytica* reappeared while patient was in the convalescent camp. He was admitted to the Orwa-el-Waska Hospital and given a second course of emetin for 12 days (one-grain injection each morning and ½ grain in keratin-coated tabloid by the mouth each night). Patient was kept in bed on milk diet. He vomited on only one occasion. The *E. histolytica* infection again disappeared and did not recur during a subsequent control of 57 days, the greater part of which was spent in the convalescent camp. The emetin courses had no effect on the patient's temperature or pulse-rate.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
1	..	++	—	++ r.b.c.	—	—	B.m.
2	E.1	—	—	+	—	—	B.uf.
3	E.1	—	—	—	—	—	B.uf.
4	E.1	—	—	—	—	—	B.uf.
5	E.1	—	—	—	—	—	B.uf.
6	E.1	—	—	—	—	—	B.uf.
7	E.1	—	—	—	—	—	B.uf.
8	E.1	—	—	—	—	—	—
9	E.1	—	—	—	—	—	B.uf.
10	E.1	—	—	—	—	—	B.uf.
11	E.1	—	—	—	—	—	—
12	E.1	—	+	+	—	—	B.uf.
13	E.1	—	+	—	—	—	B.uf.
15	..	—	—	+	—	—	B.uf.
16 C.	..	—	+++	+	++	++	B.uf.
19	..	—	+++	—	—	—	B.uf.
21	..	—	+++	+	—	—	B.uf.
23	..	—	+++	—	—	—	B.uf.
25	..	++	++	++	—	—	B.uf.
28	..	++	++	++	—	—	B.uf.
29	..	++	+	—	—	—	B.uf.
30	..	++	+	—	—	—	B.uf.
31	E.1. E.m.½. V. 1½hrs.	+++	+	+++	—	—	B.l.
32	E.1 E.m.½	+	+	—	—	—	L.b.uf.
33	E.1 E.m.½	—	+	—	—	—	B.uf.
34	E.1 E.m.½	—	+	—	+++	+++	B.l.
35	E.1 E.m.½	—	—	+	++	++	B.uf.
36	E.1 E.m.½	—	—	—	—	—	B.uf.
37	E.1 E.m.½	—	—	—	—	—	B.l.
38	E.1 E.m.½	—	+	—	—	—	B.sf.
39	E.1 E.m.½	—	—	—	—	—	B.uf.
40	E.1 E.m.½	—	—	—	—	—	L.b.uf.
41	E.1 E.m.½	—	—	—	—	—	B.f.
42	E.1 E.m.½	—	—	—	—	—	—
43	..	—	—	—	—	—	B.sf.
44	..	—	+	—	—	—	B.f.
45	..	—	+	++	—	—	B.sf.
47	..	—	+	+	—	—	B.l.
48	..	—	—	—	—	—	B.uf.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
49 C.	..	—	—	+	—	—	B.sf.
51	..	—	—	—	—	—	B.sf.
53	..	—	—	+	+++	—	B.sf.
55	..	—	+	++	+++	+++	B sf.
57	..	—	++	++	—	—	B.sf.
59	..	—	+	+	—	—	B.sf.
63	..	—	—	+++	—	—	B.sf.
66	..	—	—	+++	—	—	B.sf.
67	..	—	—	—	—	—	B.sf.
69	..	—	—	+	—	—	B.sf.
71	..	—	+	++	—	—	B.sf.
73	..	—	—	++	—	—	B.sf.
75	..	—	—	++	—	—	B.sf.
77	..	—	+	++	—	—	B.f.
79	..	—	++	+	—	—	B.sf.
81	..	—	—	++	—	—	B sf.
83	..	—	+	+	—	—	B.uf.
85	..	—	—	++	—	—	B.uf.
87	..	—	—	+	—	—	B.uf.
89	..	—	+	++	—	—	B.uf.
91	..	—	—	++	—	—	B.sf.
93	..	—	—	—	—	—	B.sf.
95	..	—	—	+++	—	—	B.sf.
97	..	—	+	+	—	—	B.sf.

Section II.

Case Lingard, H., aged 23.—Patient, who had not been abroad before, left England in September, 1914. He was on the Peninsula for eight months, where he suffered from diarrhœa. He never had dysentery. He came to Alexandria, and was found on March 20, 1916, to be a carrier of *E. histolytica* in the course of routine examination of men in Mustapha Convalescent Depot. He had large infection of cysts of *E. histolytica*, *E. coli*, and lamblia, and also I-cysts. He later had trichomonas occasionally. From March 26 he was given emetin by the mouth (one grain a day for 12 days in tinc. opii). He vomited on three occasions. He was not kept in bed, and was given chicken diet. There was no alteration in the pulse-rate, and the temperature remained normal or slightly subnormal. All the intestinal infections disappeared under the emetin, but the lamblia and trichomonas recurred soon after, and the *E. coli* later. From April 13 to April 24 the patient was given β-naphthol 15 grains three times a day for 12 days. The trichomonas did not reappear, but the lamblia and *E. coli* infections remained. After the drug was stopped, the lamblia reappeared in as great numbers as at first. There was no recurrence of the *E. histolytica* or I-cyst infections.

Days	Treatment	Findings					Stool
		E.h.c	E.c.c.	E.f.	L.c.	Trich.	
1	..	++	++	—	+++	—	B.f.
4	..	+	—	—	+++	—	B.uf.
5	..	+	+	—	+++	—	L.b.uf.
6	..	+	—	+	+++	—	B.uf.
7	E.m.1	+	—	—	+++	+	B.sf.
8	E.m.1. V. 5 min.	—	—	—	—	—	—
9	E.m.1. V. 15 min.	++	++	—	+++	—	B.f.
10	E.m.1. V. 30 min.	—	+	—	++	—	B.f.
11	E.m.1	—	—	—	+	—	B.l.
12	E.m.1	—	—	—	—	—	B.sf.
13	E.m 1	—	—	—	—	—	—
14	E.m.1	—	—	—	—	—	B.sf.
15	E.m.1	—	—	—	—	—	B.sf.
16	E.m.1	—	—	—	—	—	B.sf.
17	E.m.1	—	—	—	++	—	L.b.uf.
18	E.m.1	—	—	—	+	—	B.f.
20	..	—	—	—	+++	—	B.sf.
21	..	—	—	—	+++	—	B.sf.
22	..	—	—	—	+++	++	L.b.uf.
23	..	—	—	—	+++	—	B.uf.
24	..	—	—	—	+++	—	B.sf.
25	..	—	—	—	+++	—	B.f.
26	β-n. 15 t.d.s.	—	—	—	+++	—	B.uf.
27	β-n. 15 t.d.s.	—	—	—	—	—	—
28	β-n. 15 t.d.s.	—	—	—	—	—	—
29	β-n. 15 t.d.s.	—	—	—	—	—	B.sf.
30	β-n. 15 t.d.s.	—	—	—	—	—	—
31	β-n. 15 t.d.s.	—	—	—	—	—	—
32	β-n. 15 t.d.s.	—	—	—	—	—	B.uf.
33	β-n. 15 t.d.s.	—	+	—	++	—	L.b.uf.
34	β-n. 15 t.d.s.	—	—	—	—	—	B.uf.
35	β-n. 15 t.d.s.	—	—	—	—	—	L.b.uf.
36	β-n. 15 t.d.s.	—	—	—	—	—	L.b.uf.
37	β-n. 15 t.d.s.	—	—	—	+	—	B.uf.
38	..	—	+	+	—	—	B.uf.
39	..	—	—	—	++	—	B.uf.
40	..	—	++	—	++	—	B.uf.
42	..	—	—	—	+++	—	B.f.
45	..	—	+	—	+++	—	B.f.
47	..	—	—	—	+++	—	B.f.
50	..	—	—	—	+	—	B.f.

Case McGinty, aged 35.—Patient, who had previously been in South Africa, where he had dysentery twice, left England on September 21, 1914. He went to the Peninsula and remained there five weeks till he was invalided for dysentery. He was given no emetin. He came to Egypt and again had dysentery in Mustapha Camp. On March 24, 1916, during the routine examination of men in this camp he was found to be infected with *E. histolytica*, *E. coli* and lamblia. Later, after treatment, a tetramitus infection appeared. Patient was kept under observation till March 28, when a course of emetin (one grain a day for 12 days by the mouth) was commenced. Patient vomited a quarter of an hour after the first dose but not afterwards. After the second dose the infections of *E. histolytica*, *E. coli* and lamblia disappeared, but the *E. coli* and lamblia recurred later during the control of over one month, the last three weeks of which the patient spent in the convalescent camp. During treatment he was not kept in bed and was on a chicken diet. The emetin had no effect on the temperature or pulse-rate.

Days	Treatment	Findings							Stool
		E.h.c.	E.c.c.	E.f.	L.c.	L.f.	Tet.c.	Tet.f.	
1	..	++	++	—	+++	—	—	—	B.f.
2	..	+++	+	+	+++	—	—	—	B.sf.
3	..	+	—	+	+++	—	—	—	B.sf.
4	..	+	—	—	+++	+	—	—	B.uf.
5	E.m.1 V.15min.	+	+	—	+++	—	—	—	B.sf.
6	E.m.1	+	—	—	+++	—	—	—	B.sf.
7	E.m.1	—	—	—	—	—	—	—	—
8	E.m.1	—	—	—	—	—	—	—	B.uf.
9	E.m.1	—	—	—	—	—	—	—	B.uf.
10	E.m.1	—	—	—	—	—	—	—	B.uf.
11	E.m.1	—	—	—	—	—	—	—	B.uf.
12	E.m.1	—	—	—	—	—	—	—	B.uf.
13	E.m.1	—	—	—	—	—	—	—	B.uf.
14	E.m.1	—	—	—	—	—	—	—	B.uf.
15	E.m.1	—	—	—	—	—	—	—	B.uf.
16	E.m.1	—	—	—	—	—	—	—	B.uf.
17	..	—	—	—	—	—	—	—	B.uf.
18	..	—	—	—	++	—	—	—	B.l.
19	..	—	—	—	+++	—	+++	+++	B.uf.
20	..	—	—	—	+++	—	+++	++	B.uf.
21	..	—	—	—	++	—	++	—	B.uf.
22	..	—	—	—	+	—	++	—	B.uf.
23 C.	..	—	—	—	—	—	—	—	B.uf.
28	..	—	—	—	—	—	+	—	B.f.
31	..	—	—	—	—	—	++	+++	B.f.
36	..	—	—	—	—	—	—	—	B.f.
38	..	—	—	—	—	—	—	—	B.f.
40	..	—	—	—	—	—	—	—	B.f.
42	..	—	—	—	—	—	—	—	B.f.
44	..	—	+	+	—	—	—	+	B.f.
46	..	—	—	—	—	—	+	—	B.f.
48	..	—	—	—	—	—	—	++	B.f.

Case Howarth, R., aged 23.—Patient, who had not been abroad before, went to the Peninsula in July, 1915. He was there seven months and suffered from dysentery, for which he received 12 injections of emetin. He was invalided home, was then transferred to Egypt in January, 1916, and was found to be a carrier of *E. histolytica* on March 15 during the routine examination of men in the Mustapha Convalescent Depot. He was kept under observation till March 25, when he was given a course of emetin by the mouth (one grain a day in tinc. opii for 12 days). The patient took the emetin without vomiting and during treatment he was not kept in bed and was given chicken diet. The *E. histolytica* disappeared after the second dose and did not recur during a control of about five weeks, the last four of which were spent in the convalescent camp, where patient seems to have contracted an *E. coli* infection. The emetin had no effect on the temperature or pulse-rate.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	+++	—	—	B.f.	23	..	—	—	—	B.l.
2	..	+++	—	—	B.sf.	24	..	—	—	—	B.uf.
3	..	+++	—	+	B.sf.	25	..	—	—	—	B.uf.
4	..	++	—	+	B.uf.	26	..	—	—	—	B.l.
6	..	+	—	—	B.uf.	27	..	—	—	—	B.uf.
7	..	—	—	+++	B.l.	28	..	—	—	—	B.uf.
8	..	+++	—	+++	B.uf.	29	..	—	—	—	B.uf.
9	..	+++	—	+++	B.uf.	30	..	—	—	—	B.l.
11	E.m.1	+++	—	—	B.l.	31C.	..	—	—	—	B.uf.
12	E.m.1	++	—	—	B.sf.	32	..	—	—	—	B.f.
13	E.m.1	—	—	—	B.sf.	33	..	—	—	—	L.b.uf.
14	E.m.1	—	—	—	—	38	..	—	—	++	B.f.
15	E.m.1	—	—	—	L.b.uf.	41	..	—	—	—	B.f.
16	E.m.1	—	—	—	L.b.uf.	46	..	—	+++	—	B.f.
17	E.m.1	—	—	—	L.b.uf.	48	..	—	++	—	B.f.
18	E.m.1	—	—	—	L.b.sf.	50	..	—	++	—	B.f.
19	E.m.1	—	—	—	L.b.sf.	52	..	—	+++	—	B.f.
20	E.m.1	—	—	—	L.b.sf.	54	..	—	+++	—	B.f.
21	E.m.1	—	—	—	—	56	..	—	++	—	B.f.
22	E.m.1	—	—	—	B.uf.	58	..	—	+	—	B.f.

Case Blair, J., aged 21.—Patient, who had been three years in Australia, left there in February, 1915, and came direct to Egypt, whence he was transferred to the Peninsula. He remained there till July, when he was invalided to Egypt for deafness. In February, 1916, while in Cairo he had his first attack of dysentery, for which he went into hospital. He was given no emetin. He returned to duty, but again had dysentery in March, and was then treated with emetin (two injections a day for a week). He had another attack while in convalescent camp. He was again ill with dysentery in Cairo and was given a further 14 injections of emetin, and was a second time sent to Montazah Convalescent Camp at Alexandria, where dysentery again recurred. He was admitted to the Orwa-el-Waska Hospital on June 6, 1916, and was found to be passing stools with blood and mucus containing large numbers of active amœbæ with included red blood corpuscles. From June 7 onwards, patient was given a 12-day course of methyl emetin sulphate (one grain injection each morning and one grain in keratin-coated tabloid by the mouth at night). This course of methyl emetin produced no vomiting, but had no effect on the infection. Accordingly the injections were stopped and emetin hydrochloride in keratin-coated tabloid (one grain) was given each night for 12 nights instead of the methyl emetin sulphate. Though this change of drug was made without the patient's knowledge, he vomited on the first three nights after taking the drug. The *E. histolytica* infection disappeared after the fourth day of treatment, but recurred again later when cysts of the amœba were found for the first time. The emetin courses had no effect on the patient's temperature or pulse-rate. During treatment he was confined to bed and was on milk diet.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	—	—	+++r.b.c.	B.l. B.m.	16	E.m.1. V. ½ hr.	—	—	+	B.uf.
2	M.E.2	—	—	+++r.b.c.	B.l. B.m.						
3	M.E.2	—	—	+	B.uf.	17	E.m.1	—	—	++	B.uf.
4	M.E.2	—	—	—	—	18	E.m.1	—	—	—	B.uf.
5	M.E.2	—	—	—	—	19	E.m.1	—	—	—	B.uf.
6	M.E.2	—	—	—	B.uf.	20	E.m.1	—	—	—	B.uf.
7	M.E.2	—	—	+++	B.uf.	21	E.m.1	—	—	—	B.uf.
8	M.E.2	—	—	+++	B.uf.	22	E.m.1	—	—	—	B.uf.
9	M.E.2	—	—	—	B.uf.	23	E.m.1	—	—	—	B.uf.
10	M.E.2	—	—	—	B.uf.	24	E.m.1	—	—	—	—
11	M.E.2	—	—	++ r.b.c.	B.uf. B.m.	25	E.m.1	—	—	—	B.uf.
12	M.E.2	—	—	++ r.b.c.	B uf. B.m.	26	..	—	—	+	B.uf.
13	M.E.2	—	—	+	B.uf.	27	..	—	—	—	B.uf.
14	E.m.1. V. ½ hr.	—	—	+	B.uf.	28	..	—	—	—	B.uf.
						30C.	..	—	—	+	B.uf.
15	E.m.1. V. inst.	—	—	+	B.uf.	35	..	—	—	—	B.uf.
						39	..	+++	—	—	B.uf.
						43	..	+++	++	++	B.uf.

Section III.

Case Lyall, aged 28.—Patient, who had not been abroad before, left England in May, 1915, and went to the Peninsula, where he remained six months. He was then transferred to Egypt, where, on April 26, during the routine examination of men in Mustapha Camp, he was found to be a carrier of *E. histolytica*. He had also an infection of *E. nana*. There was no history of dysentery. Patient was given a 12-day course of emetin. From May 2 (one grain injection each morning, and ½ grain in keratin-coated tabloid by the mouth each night). During the treatment patient was kept in bed on milk diet. There was no vomiting. The *E. histolytica* cysts disappeared from the stool, and did not recur during a control of over one month, the greater part of which time patient spent in the convalescent camp on light duty. The emetin had no effect on patient's temperature or pulse-rate. The *E. nana* infection also disappeared during the emetin course, and did not recur.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	E.n.c.	E.n.f.				E.h.c.	E.f.	E.n.c.	E.n.f.	
1	..	+++	—	—	—	B.uf.	20	..	—	—	—	—	L.b.uf.
4	..	+++	—	—	++	B.uf.	21	..	—	—	—	—	B.f.
6	..	++	+	+++	+++	B.sf.	22	..	—	—	—	—	B.f.
7	E.1 E.m.½	++	—	+++	+++	B.l.	23	..	—	—	—	—	L.b.l.
8	E.1 E.m.½	+	—	+++	—	B.l.	25	..	—	—	—	—	L.b.uf.
9	E.1 E.m.½	—	—	—	—	B.uf.	27 C.	..	—	—	—	—	B.uf.
10	E.1 E.m.½	—	—	—	—	L.b.l.	29	..	—	—	—	—	B.f.
11	E.1 E.m.½	—	—	—	—	B.f.	31	..	—	—	—	—	B.f.
12	E.1 E.m.½	—	—	—	—	L.b.sf.	33	..	—	—	—	—	B.f.
13	E.1 E.m.½	—	—	—	—	L.b.uf.	35	..	—	—	—	—	B.uf.
14	E.1 E.m.½	—	—	—	—	L.b.uf.	39	..	—	—	—	—	B.sf.
15	E.1 E.m.½	—	—	—	—	—	47	..	—	—	—	—	B.sf.
16	E.1 E.m.½	—	—	—	—	B.uf.	53	..	—	—	—	—	B.sf.
17	E.1 E.m.½	—	—	—	—	L.b.sf.	55	..	—	—	—	—	B.sf.
18	E.1 E.m.½	—	—	—	—	B.sf.	57	..	—	—	—	—	B.sf.
19	..	—	—	—	—	B.uf.	59	..	—	—	—	—	B.sf.

Case Palmer, aged 26.—Patient, who had never been abroad before, left England in February, 1916, and came direct to Egypt. On April 30, during the routine examination of men in Mustapha Camp, he was found to be a carrier of *E. histolytica*. He had also an infection of *E. coli*, lamblia, and tetramitus. There was no history of dysentery. Patient was kept under observation till March 6, when he was given a course of emetin for 12 days (one grain injection each morning, and ½ grain in keratin-coated tabloid each night). Patient vomited on the second day after taking the tabloid, but not subsequently. During treatment he was kept in bed on milk diet. All the infections disappeared during treatment, but the *E. coli* and tetramitus returned soon after. There was no return of the *E. histolytica* or lamblia infection during a control of over one month, the last three weeks of which patient spent in the convalescent camp, where he performed light duty. The emetin had no influence on the patient's temperature or pulse-rate.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	L.c.	Tet.c.	Tet.f.	
1	..	++	++	+	—	++	++	B.uf.
3	..	+	++	—	—	—	—	B.f.
4	..	+++	++	—	—	—	—	B.f.
5	..	+++	+	—	++	++	—	L.b.uf.
6	..	+	+	—	++	—	—	L.b.uf.
7	E.1 E.m.½	+	—	—	+++	—	—	B.uf.
8	E.1. E.m.½. V. 2 hrs.	—	++	—	+++	—	—	L.b.uf.
9	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	L.c.	Tet.c.	Tet.f.	
10	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
11	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
12	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
13	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
14	E.1 E.m.½	—	—	—	—	—	—	L.b.sf.
15	E.1 E.m.½	—	—	—	—	—	—	B.l.
16	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
17	E.1 E.m.½	—	—	—	—	—	—	B.uf.
18	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
19	..	—	—	—	—	—	++	L.b.uf.
20	..	—	—	—	—	—	—	B.uf.
21	..	—	+	—	—	+++	—	B.uf.
22	..	—	+	—	—	+++	+	B.uf.
23	..	—	++	—	—	++	—	L.b.uf.
24 C.	..	—	++	—	—	++	—	B.uf.
26	..	—	+	—	—	—	+++	B.uf.
28	..	—	++	—	—	+++	+	B.uf.
30	..	—	+	+	—	++	+++	B.uf.
32	..	—	++	—	—	++	+++	B.uf.
34	..	—	+++	+	—	—	+++	B.uf.
36	..	—	+++	+	—	—	—	B.sf.
38	..	—	+++	—	—	—	—	B.sf.
40	..	—	+++	+	—	—	—	B.sf.
42	..	—	++	—	—	—	—	B.sf.
44	..	—	+	—	—	—	—	B.sf.
46	..	—	++	+	—	—	—	B.sf.
48	..	—	+	+	—	—	—	B.sf.
50	..	—	—	++	—	—	+++	B.sf.
52	..	—	++	++	—	—	—	B.sf.

Case English, H., aged 19.—Patient, who had not been abroad before, left England in October, 1915, for the Peninsula, where he remained two months before being transferred to Egypt. On May 3, 1916, he was found to be a carrier of *E. histolytica* during the routine examination of cooks in Metras Camp. Patient gave no history of dysentery. There was also an infection of *E. coli* and *E. nana*. Patient was observed till May 9 when a 12-day course of emetin was commenced (one-grain injection in the morning and ½ grain in keratin-coated tabloid by the mouth at night). During treatment patient was kept in bed on milk diet. Patient vomited on five occasions after taking the emetin, but notwithstanding this the *E. histolytica* as well as the *E. coli* and *E. nana* infections disappeared, and the only infection to recur was that of *E. nana* during a control of over one month, the greater part of which patient spent in the convalescent camp on light duty. The emetin had no appreciable effect on the patient's temperature or pulse-rate.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
1	..	++	—	+	++	—	B.uf.
3	..	++	+	—	—	++	B.uf.
4	..	+++	—	+++	—	—	B.l.
5	..	++	+	—	++	++	B.l.
6	..	+	—	+	+++	+++	B.l.
7	E.1 E.m.½	+	—	+++	+++	+++	B.uf.
8	E.1 E.m.½. V.2½hrs.	—	—	—	—	—	—
9	E.1 E.m.½	—	—	—	—	—	—
10	E.1 E.m.½	—	—	—	++	++	B.l.
11	E.1 E.m.½. V.1½hrs.	—	—	—	—	—	B.uf.
12	E.1 E.m.½	—	—	—	—	—	B.uf.
13	E.1 E.m.½	—	—	—	—	—	B.uf.
14	E.1 E.m.½. V.½ hr.	—	—	—	—	—	L.b.uf.
15	E.1 E.m.½. V.1½hrs.	—	—	—	—	—	L.b.uf.
16	E.1 E.m.½	—	—	—	—	—	L.b.uf.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	
17	E.1 E.m.½. V.1¼hrs.	—	—	—	—	—	L.b.uf.
18	E.1 E.m.½	—	—	—	—	—	B.l.
19	..	—	—	—	—	—	L.b.sf.
20	..	—	—	—	—	—	B.l.
21	..	—	—	—	—	—	B.sf.
22	..	—	—	—	—	—	B.uf.
23	..	—	—	—	—	—	B.uf.
24 C.	..	—	—	—	—	—	—
25	..	—	—	—	—	—	B.f.
27	..	—	—	—	—	—	B.uf.
29	..	—	—	—	+++	+	B.uf.
31	..	—	—	—	+++	++	B.uf.
33	..	—	—	—	++	++	B.sf.
35	..	—	—	—	—	—	B.sf.
37	..	—	—	—	—	—	B.sf.
39	..	—	—	—	—	—	B.sf.
41	..	—	—	—	—	—	B.sf.
43	..	—	—	—	—	—	B.sf.
45	..	—	—	—	++	++	B.f.
47	..	—	—	—	+++	+++	B.sf.
51	..	—	—	—	—	—	B.uf.

Case Howard, J., aged 32.—Patient, who had not been abroad before, left England on March 18, 1915 and came direct to Egypt, where he remained. On May 1, 1916, during the routine examination of men in Mustapha Camp, patient was found to be a carrier of *E. histolytica*. There was also an infection, *E. coli*, tetramitus, trichomonas and I-cysts. There was no history of dysentery. Patient was kept under observation till May 9 and was then given a course of emetin for 12 days (one-grain injection each morning and ½ grain keratin-coated tabloid by the mouth each night). The patient was kept in bed on milk diet during the treatment. There was no vomiting. The *E. histolytica* quickly disappeared but not the tetramitus or trichomonas. There was no recurrence of the *E. histolytica* infection during a control of one month, the greater part of which patient spent in the convalescent camp on light duty. The course of emetin had no effect on the patient's temperature or pulse-rate.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	Trich.	
1	..	+	+	—	—	—	—	B.uf.
5	..	—	—	—	—	++	+	B.uf.
6	..	+	—	—	—	+++	+	B.uf.
7	..	—	—	+	+++	+++	+++	B.uf.
8	..	—	—	—	—	—	—	B.sf.
9	E.1 E.m.½	++	—	++	—	—	—	B.uf.
10	E.1 E.m.½	—	—	++	—	+	++	B.uf.
11	E.1 E.m.½	—	—	—	+++	++	+++	B.uf.
12	E.1 E.m.½	—	—	—	—	—	—	—
13	E.1 E.m.½	—	—	—	—	++	+++	B.l.
14	E.1 E.m.½	—	—	—	—	++	++	B.l.
15	E.1 E.m.½	—	—	—	—	—	—	B.l.
16	E.1 E.m.½	—	—	—	+	—	+	L.b.uf.
17	E.1 E.m.½	—	—	—	++	++	+++	B.uf.
18	E.1 E.m.½	—	—	—	—	+++	+++	L.b.uf.
19	E.1 E.m.½	—	—	—	—	+++	+++	L.b.uf.
20	E.1 E.m.½	—	—	—	—	++	+++	L.b.uf.
21	..	—	—	—	—	++	++	L.b.uf.
22	..	—	—	—	—	+++	+++	B.uf.
23 C.	..	—	—	—	++	+++	+++	B.uf.
24	..	—	—	—	++	+++	+++	B.uf.
25	..	—	—	—	++	+++	+++	B.uf.
27	..	—	++	+	+++	+++	+++	B.uf.
29	..	—	++	+++	++	+++	—	B.uf.
31	..	—	++	++	+++	+++	—	B.uf.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	Trich.	
33	..	—	+++	++	—	+++	—	B.sf.
35	..	—	+++	+++	—	+	—	B.uf.
37	..	—	+++	++	+	+	—	B.sf.
39	..	—	++	++	+	++	+	B.sf.
41	..	—	—	+++	+	+	—	B.sf.
43	..	—	+	+	—	+	—	B.sf.
45	..	—	++	+	—	—	—	B.sf.
47	..	—	+	+	—	+	—	B.sf.
49	..	—	++	++	—	—	—	B.sf.
51	..	—	+	+	—	+	—	B.sf.

Case Hyde, aged 20.—Patient left England in September, 1915, and went direct to the Peninsula, where he remained five weeks. He was wounded and invalided to Egypt. On April 25, 1916, during the routine examination of men in Mustapha Camp he was found to be a carrier of *E. histolytica*. He had also infection of trichomonas and later tetramitus and *E. nana*. There was no history of dysentery. Patient was kept under observation till May 3, when a 12-day course of emetin was commenced (one grain injection in the morning and ½ grain in keratin-coated tabloid by the mouth each night). During treatment patient was kept in bed on milk diet. Patient vomited on five occasions after taking the emetin. The *E. histolytica* cysts vanished from the stool after the first dose and did not recur during a control of over one month, three weeks of which patient spent in the convalescent camp, where he performed light duty. The course of emetin produced no change in the temperature or pulse-rate. There developed, however, a stomatitis with aphthæ on the tonsils, lips and tongue. This persisted during the treatment but disappeared soon after.

Days	Treatment	Findings						Stool
		E.h.c.	E.f.	E.n.c.	E.n.f.	Tet.f.	Trich.	
1	..	++	—	—	—	—	—	B.uf.
5	..	+	—	—	—	—	—	B.uf.
6	..	++	++	—	—	—	+++	B.uf.
7	..	++	+	—	—	—	+++	B.uf.
8	..	++	—	—	—	—	++	B.uf.
9	E.1 E.m.½. V. 1 hr.	++	—	—	—	—	—	B.uf.
10	E.1 E.m.½	—	—	—	—	—	—	B.uf.
11	E.1 E.m.½. V. ¾ hr.	—	—	—	—	—	—	B.l.
12	E.1 E.m.½	—	—	—	—	—	—	B.uf.
13	E.1 E.m.½. V. 1¾ hr.	—	—	—	—	—	—	B.l.
14	E.1 E.m.½	—	—	++	++	—	—	B.l.
15	E.1 E.m.½	—	—	—	—	—	—	B.l.
16	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
17	E.1 E.m ½	—	—	—	—	—	—	L.b.uf.
18	E.1 E.m.½. V. 1 hr.	—	—	—	—	—	—	B.sf.
19	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
20	E.1 E.m.½	—	—	—	—	—	—	B.uf.
21	..	—	—	—	—	—	—	B.uf.
22	..	—	—	—	—	—	—	B.l.
23	..	—	—	—	—	—	—	B.l.
24	..	—	—	—	—	—	—	B.l.
25	..	—	—	—	—	—	+++	B.l.
26	..	—	—	—	—	—	—	B.uf.
27	..	—	—	—	—	+++	++	B.l.
28 C.	..	—	—	—	—	+++	—	L.b.l.
30	..	—	—	—	—	+++	—	B.f.
32	..	—	—	—	—	—	+++	B.f.
34	..	—	—	—	—	—	++	B.uf.
36	..	—	—	—	—	—	+	B.uf.
41	..	—	—	+	+	—	—	B.sf.
52	..	—	—	—	—	—	—	B.sf.
54	..	—	—	—	—	—	—	B.sf.
56	..	—	—	—	—	—	—	B.sf.
58	..	—	—	—	—	—	—	B.sf.
60	..	—	—	—	—	—	—	B.sf.

Case Cherill, W., aged 20.—Patient, who had not been abroad before, left England in August, 1915, for the Peninsula, where he remained four months. He was transferred to Egypt in December, where on April 22, 1916, he was found to be a carrier of *E. histolytica* during the routine examination of men in Mustapha Camp. There were also infections of *E. coli* and I-cysts and an irregular infection of *E. nana* and lamblia. There was no history of dysentery. Patient was kept under observation till May 2, when a course of emetin was commenced (one grain injection in the morning and ½ grain in keratin-coated tabloid at night for 12 days). Patient, who was kept in bed and given milk diet, vomited only once on the third night of treatment. All the infections disappeared and none recurred during a control of over one month except the *E. coli* and *E. nana*. The last three weeks of control patient spent in the convalescent camp where he performed light duty. The emetin had no effect on the patient's temperature or pulse-rate.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	L.c.	E.n.c.	E.n.f.	
1	..	+++	+++	+	+	—	—	B.uf.
4	..	—	+	+	—	—	+	B.sf.
5	..	—	+	++	—	—	+	B.uf.
6	..	+	+	—	—	—	—	B.uf.
7	..	—	—	+++	—	—	—	B.l.
8	..	++	++	—	++	—	+++	B.l.
9	..	++	+	—	—	—	+	B.uf.
10	..	++	++	—	—	++	—	B.sf.
11	E.1 E.m.½	+++	+++	—	+	++	++	B.uf.
12	E.1 E.m.½	+++	+++	—	—	—	—	L.b.uf.
13	E.1 E.m.½. V. 1 hr.	+	++	—	—	—	—	L.b.sf.
14	E.1 E.m.½	—	+	—	—	—	—	B.l.
15	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
16	E.1 E.m.½	—	—	—	—	—	—	B.uf.
17	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
18	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
19	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
20	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
21	E.1 E.m.½	—	—	—	—	—	—	—
22	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
23	..	—	—	—	—	—	—	B.sf.
24	..	—	—	—	—	—	—	B.uf.
25	..	—	—	—	—	—	—	B.l.
26	..	—	—	—	—	—	—	B.uf.
27	..	—	—	—	—	—	—	L.b.uf.
29	..	—	—	—	—	—	—	L.b.uf.
31 C.	..	—	—	—	—	—	—	B.f.
34	..	—	—	—	—	—	—	B.f.
36	..	—	++	+	—	++	++	B.sf.
38	..	—	+	++	—	—	—	B.sf.
40	..	—	+	+	—	—	—	B.sf.
42	..	—	+++	++	—	—	—	B.sf.
44	..	—	++	+	—	++	++	B.sf.
46	..	—	++	+	—	—	—	B.sf.
48	..	—	++	++	—	—	—	B.sf.
50	..	—	++	++	—	—	—	B.sf.
52	..	—	+	—	—	—	—	B.l.
54	..	—	—	++	—	—	—	B.sf.

Case Wing, A., aged 22.—Patient, who had never been abroad before, served in France from June to December, 1915. He was then transferred to Egypt, where he arrived on January 5, 1916. On April 26 patient was admitted to hospital with diarrhœa. The stool was found to consist of thick tenacious mucus mixed with fæcal matter, but without blood. Microscopically the mucus was impregnated with myriads of free lamblia, while the fæcal matter showed numerous free and encysted forms as well as small free amœbæ. The next day patient passed

a light brown liquid stool without mucus ; lamblia cysts were present in large numbers, as well as small free amœbæ and cysts of *E. histolytica*. It was in this case that the dividing forms of lamblia described elsewhere were found. The presence of the lamblia in such enormous numbers in the mucus and the fact that *E. histolytica* were in the "minuta" and encysted stages would suggest that the attack was not one of amœbic dysentery but rather of lamblia irritation. Apparently the case was a carrier of *E. histolytica* in which symptoms due to overwhelming lamblia infection developed. Patient had not been ill with diarrhœa or dysentery before. He was kept under observation till May 2 and then given a 12-day course of emetin (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth at night). During the treatment he was kept in bed on milk diet. The *E. histolytica* and lamblia infections disappeared, but infections of *E. coli* and *E. nana* became evident. The *E. histolytica* infection did not recur during a control of over six weeks, part of which patient spent in convalescent camp on light duty. The lamblia infection recurred soon after the emetin course was finished and persisted till patient developed jaundice, for which he was re-admitted to hospital. Curiously enough, the lamblia diminished in numbers two days before patient was admitted for jaundice and were completely absent when the characteristic jaundice stools were found. During the attack the *E. coli* and *E. nana* infections were also absent. The lamblia infection recurred a fortnight after the jaundice had disappeared. The emetin course had no effect on the patient's temperature or pulse-rate.

Days	Treatment	Findings									Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	L.c.	L.f.	Tet.c.	Tet.f.	
1	..	—	—	++	—	—	+++	+++	—	—	L.b.f.m.
3	..	+	—	++	—	—	+++	—	—	—	L.b.l.
4	..	—	—	+	—	—	++	—	—	—	B.l.
5	..	+	—	—	—	—	++	—	—	—	B.uf.
6	..	—	—	+	—	+++	++	—	—	—	B.l.
7	E.1 E.m.½. V.¾ hr.	—	+	—	—	++	++	—	—	—	L.b.uf.
8	E.1 E.m.½	—	++	—	—	—	+++	—	—	—	L.b.l.
9	E.1 E.m.½	—	—	+	—	—	—	—	—	—	L.b.l.
10	E.1 E.m.½	—	—	—	—	—	—	—	—	—	L.b.uf.
11	E.1 E.m.½	—	—	—	+++	+++	—	—	—	—	L.b.uf.
12	E.1 E.m.½	—	—	—	++	++	—	—	—	—	B.uf.
13	E.1 E.m.½	—	—	+++	—	++	—	—	—	—	L.b.uf.
14	E.1 E.m.½	—	—	—	—	—	—	—	—	—	—
15	E.1 E.m.½	—	—	—	—	—	—	—	—	—	—
16	E.1 E.m.½	—	+	+	—	—	—	—	—	—	L.b.uf.
17	E.1 E.m.½	—	—	—	—	—	—	—	—	—	L.b.l.
18	E.1 E.m.½	—	—	—	—	—	—	—	—	—	—
19	..	—	—	—	—	—	—	—	—	—	L.b.l.
20	..	—	—	—	—	—	++	—	—	—	L.b.uf.
21	..	—	—	—	—	—	++	—	—	—	B.uf.
22	..	—	—	—	—	—	+++	—	—	—	L.b.uf.
23	..	—	—	—	—	—	+++	—	—	—	L.b.uf.
25	..	—	—	—	—	—	+++	—	—	—	L.b.uf.
27	..	—	++	—	—	—	+++	—	—	—	B.uf.
29	..	—	+	—	++	++	+++	—	—	—	B.uf.
30 C.	..	—	—	—	++	++	++	—	—	—	B.uf.
32	..	—	++	—	+	+	++	—	—	—	B.uf.
34	..	—	+	—	—	—	+	—	—	—	B.uf.
36	..	—	++	—	—	—	++	—	—	—	B.uf.
40	..	—	+	—	—	—	+	—	—	—	L.b.sf.
42	..	—	—	—	—	—	—	—	—	—	Y.w.sf.
43	..	—	—	—	—	—	—	—	—	—	Y.w.sf.
44	..	—	—	—	—	—	—	—	—	—	Y.w.sf.
46	..	—	—	—	—	—	—	—	—	—	Y.w.sf.
47	..	—	—	—	—	—	—	—	—	—	Y.w.sf.
49	..	—	+	—	+++	+++	—	—	—	—	B.uf.
50	..	—	++	—	+++	+++	—	—	—	—	B.uf.

Days	Treatment	Findings									Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	L.c.	L.f.	Tet.c.	Tet.f.	
51	..	—	++	—	+++	+++	—	—	—	—	B.uf.
52	..	—	++	—	+++	+++	—	—	—	—	B.uf.
53	..	—	++	—	++	++	—	—	—	—	B.uf.
54	..	—	++	+	++	++	—	—	—	—	B.uf.
55	..	—	+	—	++	++	—	—	—	—	B.f.
56	..	—	+	+	++	++	—	—	—	—	B.f.
57	..	—	—	—	+++	+++	—	—	—	—	B.f.
58	..	—	—	—	++	++	—	—	—	—	B.f.
59	..	—	+	—	+	+	—	—	—	—	B.uf.
60	..	—	—	—	+	—	—	—	—	—	B.uf.
61	..	—	—	—	—	—	—	—	—	—	B.uf.
62	..	—	—	—	—	—	—	—	—	—	B.uf.
63	..	—	—	—	—	—	—	—	—	—	B.uf.
64	..	—	—	—	++	++	++	—	—	—	B.f.
65	..	—	+	+	—	—	++	—	++	++	B.f.

Case Sargeant, A., aged 22.—Patient, who had not been abroad before, left England in June, 1915, and was four months on the Peninsula. He was then transferred to Egypt, where, on April 26, during the routine examination of men in Mustapha Camp, he was found to be a carrier of *E. histolytica*. There was also an infection of *E. coli*, tetramitus, and lamblia, while later on an infection of *E. nana* appeared. There was no history of dysentery. Patient was kept under observation till May 2, when a course of emetin for 12 days was commenced (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth each night). During treatment patient was kept in bed on milk diet. Patient vomited on two occasions after taking the tabloid. All the infections disappeared during the treatment, but the tetramitus and *E. coli* infections recurred. There was no recurrence of the *E. histolytica* infection during a control of over one month, three weeks of which patient spent in the convalescent camp on light duty. The emetin had no effect on patient's temperature, but the pulse-rate was slightly increased towards the end of and after the course of treatment.

Days	Treatment	Findings								Stool
		E.h.c.	E.c.c.	E.f.	L.c.	Tet.c.	Tet.f.	E.n.c.	E.n.f.	
1	..	+	+++	+	—	+	—	—	—	B.uf.
4	..	++	+	—	++	—	+	—	—	B.uf.
5	..	+	—	—	+++	—	+++	—	—	B.l.
7	E.1 E.m.½. V. ¾ hr.	—	—	—	—	—	—	—	—	—
8	E.1 E.m.½	++	+	—	—	+++	+++	—	—	B.uf.
9	E.1 E m.½	+	—	—	—	—	—	—	—	B.uf.
10	E.1 E.m.½	—	—	—	—	—	—	—	—	—
11	E.1 E.m.½	—	—	—	—	—	++	—	—	B.uf.
12	E.1 E.m.½	—	—	—	—	—	—	—	—	—
13	E.1 E.m.½	—	—	—	—	+++	—	—	—	B.sf.
14	E.1 E.m.½	—	—	—	—	—	—	—	—	L.b.uf.
15	E.1 E.m.½. V. 1½ hrs.	—	—	—	—	—	—	—	—	B.sf.
16	E.1 E.m.½	—	—	—	—	—	—	—	—	B.l.
17	E.1 E.m.½	—	—	—	—	—	—	—	—	L.b.uf.
18	E.1 E.m.½	—	—	—	—	—	—	—	—	—
19	..	—	—	—	—	—	—	—	—	L.b.uf.
20	..	—	—	—	—	—	—	—	—	B.l.
21	..	—	—	—	—	—	—	—	—	B.uf.
22	..	—	—	+	—	—	+++	—	—	B.uf.
23	..	—	+	+	—	—	+++	—	—	B.uf.
24	..	—	+	++	—	+++	+++	++	++	B.uf.
25	..	—	+	—	—	+++	+++	++	++	B.sf.
26	..	—	++	—	—	++	—	++	++	B.uf.
27	..	—	++	+	—	—	+++	—	—	B.uf.
28 C.	..	—	++	—	—	++	—	—	—	B.sf.
30	..	—	+	—	—	—	—	—	—	B.f.
32	..	—	+	—	—	—	—	—	—	B.f.
34	..	—	+	—	—	—	—	—	—	B.uf.
36	..	—	+	—	—	—	—	—	—	B.uf.
38	..	—	+	—	—	—	—	—	—	B.uf.

Days	Treatment	Findings								Stool
		E.h.c.	E.c.c.	E.f.	L.c.	Tet.c.	Tet.f.	E.n.c.	E.n.f.	
40	..	—	+	—	—	—	—	—	—	B.sf.
42	..	—	+	—	—	—	—	—	—	B.sf.
44	..	—	+	—	—	—	—	—	—	B.sf.
46	..	—	++	++	—	+	+	++	++	B.sf.
48	..	—	+	—	—	—	—	—	—	B.sf.
50	..	—	+	—	—	—	—	—	—	B.sf.

Case Baker, aged 20.—Patient, who had not been abroad before, left England in April, 1915, and came direct to Egypt, where he remained. On April 4, and again on April 24, 1916, he was found to be a carrier of *E. histolytica* during the routine examination of cooks in Mustapha Camp. He gave no history of dysentery, but had had attacks of diarrhœa. Patient was given a course of emetin for 12 days from May 2 (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth each night). Patient vomited half an hour after the first dose, but not afterwards. During treatment he was kept in bed and given milk diet. The *E. histolytica* infection, which was not evident in the stool the day before treatment was commenced, did not recur during a control of over one month after treatment. For the last three weeks of the observation patient was in convalescent camp, where he performed light duty. A large infection of lamblia, which had not occurred before, appeared soon after the course of emetin was completed, and towards the end of the observation an infection of *E. coli*. The emetin had no effect on the temperature or pulse-rate.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	L.c.	L.f.	
1	..	+	—	—	—	—	B.f.
7	..	++	—	—	—	—	B.f.
9	..	++	—	—	—	—	B.uf.
11	..	—	—	—	—	—	B.uf.
12	E.1 E.m.½. V. ½ hr.	—	—	—	—	—	—
13	E.1 E.m.½	—	—	—	—	—	B.l.
14	E.1 E.m.½	—	—	—	—	—	B.sf.
15	E.1 E.m.½	—	—	—	—	—	L.b.uf.
16	E.1 E.m.½	—	—	—	—	—	B.l.
17	E.1 E.m.½	—	—	—	—	—	B.sf.
18	E.1 E.m.½	—	—	—	—	—	—
19	E.1 E.m.½	—	—	—	—	—	B.f.
20	E.1 E.m.½	—	—	—	—	—	B.uf.
21	E.1 E.m.½	—	—	—	—	—	L.b.uf.
22	E.1 E.m.½	—	—	—	—	—	L.b.l.m.
23	E.1 E.m.½	—	—	—	—	—	L.b.uf.
24	..	—	—	—	—	—	B.f.
26	..	—	—	—	+++	—	B.f.
27	..	—	—	—	+++	—	B.l.
28	..	—	—	—	+++	+++	B.uf.
30	..	—	—	—	+++	—	B.f.
32 C.	..	—	—	—	+++	—	B.f.
35	..	—	—	—	+++	—	B.f.
37	..	—	—	—	+++	—	B.f.
39	..	—	++	—	+++	—	B.uf.
43	..	—	+	+	—	—	B.uf.
47	..	—	+	—	—	—	B.uf.
49	..	—	+	+	—	—	B.uf.
53	..	—	+++	—	+	—	B.sf.
55	..	—	++	—	++	—	B.sf.

Case Graham, C., aged 42.—Patient, who had not been abroad before, left England in August, 1915, and went to the Peninsula, where he remained till the end of the year, when he was transferred to Egypt. On April 25, 1916, during the routine examination of cooks in

Mustapha Camp he was found to be a carrier of *E. histolytica*. He gave no history of dysentery. On the first examination there were present also infections of *E. coli* and I-cysts, but these were not found two days later and they did not reappear during the time patient was under observation. He was given a course of emetin for 12 days (one grain injection each morning and ½ grain in keratin-coated tabloid at night). Patient was kept in bed and on milk diet during the treatment. There was no vomiting. The *E. histolytica* infection disappeared and did not recur during a control of one month, three weeks of which patient spent in the convalescent camp, where he performed light duty. The emetin had no effect on the patient's temperature or pulse-rate. During the second half of the period of observation there were present in the stool on a few occasions cysts of tetramitus and there developed a large infection of *E. nana*.

Days	Treatment	Findings						Stool
		E.h.c.	E.c.c.	E.f.	E.n.c.	E.n.f.	Tet.c.	
1	..	++	+	—	—	—	—	B.uf.
3	..	+++	—	+	++	++	—	B.uf.
4	..	+++	—	—	—	—	—	B.uf.
5	..	+++	—	—	—	—	—	B.f.
6	..	+++	—	—	—	—	—	B.f.
7	..	+++	—	—	—	—	—	B.sf.
8	E.1 E.m.½	+++	—	—	—	—	—	B.sf.
9	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
10	E.1 E.m.½	+	—	—	—	—	—	L.b.uf.
11	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
12	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
13	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
14	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
15	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
16	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
17	E.1 E.m.½	—	—	—	—	—	—	L.b.uf.
18	E.1 E.m.½	—	—	—	—	—	—	L.b.sf.
19	E.1 E.m.½	—	—	—	—	—	—	L.b.l.
20	..	—	—	—	—	—	—	L.b.sf.
21	..	—	—	—	—	—	—	L.b.uf.
22	..	—	—	—	—	—	—	L.b.f.
23	..	—	—	—	—	—	—	B.uf.
24	..	—	—	—	+++	—	—	B.sf.
25	..	—	—	—	+++	—	—	B.sf.
26	..	—	—	—	+++	—	+	L.b.uf
27	..	—	—	—	+++	—	+	B.f.
28 C.	..	—	—	—	+++	—	+	B.f.
31	..	—	—	—	+++	—	+	B.l.
33	..	—	—	—	+++	+++	—	B.uf.
35	..	—	—	—	++	++	—	B.uf.
37	..	—	—	—	+++	++	—	B.uf.
39	..	—	—	—	+++	+++	—	B.sf.
41	..	—	—	—	+	+	—	B.sf.
43	..	—	—	—	+++	+++	—	B.sf.
45	..	—	—	—	+++	+++	—	B.sf.
47	..	—	—	—	+++	+++	—	B.sf.
49	..	—	—	—	+++	+++	—	B.sf.
51	..	—	—	—	+++	+++	—	B.sf.

Case Carr, P., aged 33.—Patient, who had served in South Africa, left England in April, 1916, and came direct to Egypt, where, on May 19, during the routine examination of men in Mustapha Camp he was found to be a carrier of *E. histolytica*. There was no history of previous dysentery. Patient was kept under observation till May 30, during which time he continued to pass cysts and amœbæ in very large numbers. On May 30, a 12-day course of emetin was commenced (one grain injection each morning and ½ grain in keratin-coated tabloid each evening). During treatment he was kept in bed on milk diet. There was no vomiting. In spite of the magnitude of the infection there was complete disappearance of the

cysts and amœbæ after the first day of treatment and there was no recurrence during a control of over one month, three weeks of which patient spent in the convalescent camp on light duty. The emetin course did not affect the patient's temperature or pulse-rate. There was an infection of *E. nana*, which was only observed before the treatment.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	E.n.c.	E.n.f.				E.h.c.	E.f.	E.n.c.	E.n.f.	
1	..	+++	++	++	++	B.uf.	22	E.1 E.m.½	—	—	—	—	B.f.
3	..	+++	—	+	+	B.sf.	23	E.1 E.m.½	—	—	—	—	B.uf.
4	..	+	+++	—	—	B.sf.	24	..	—	—	—	—	B.uf.
5	..	+++	—	—	—	B.sf.	26	..	—	—	—	—	B.sf.
6	..	+++	+++	++	++	B.sf.	28 C.	..	—	—	—	—	B.sf.
7	..	+++	++	+++	+++	B.sf.	29	..	—	—	—	—	B.sf.
10	..	+++	++	—	—	B.sf.	31	..	—	—	—	—	B.sf.
11	..	+++	++	—	—	B.uf.	33	..	—	—	—	—	B.sf.
12	E.1 E.m.½	+++	+	—	—	B.uf.	35	..	—	—	—	—	B.sf.
13	E.1 E.m.½	—	—	—	—	B.l.	37	..	—	—	—	—	B.sf.
14	E.1 E.m.½	—	—	—	—	B.uf.	39	..	—	—	—	—	B.sf.
15	E.1 E.m.½	—	—	—	—	B.uf.	42	..	—	—	—	—	B.sf.
16	E.1 E.m.½	—	—	—	—	B.uf.	43	..	—	—	—	—	B.sf.
17	E.1 E.m.½	—	—	—	—	B.uf.	45	..	—	—	—	—	B.sf.
18	E.1 E.m.½	—	—	—	—	—	47	..	—	—	—	—	B.sf.
19	E.1 E.m.½	—	—	—	—	L.b.sf.	49	..	—	—	—	—	B.sf.
20	E.1 E.m.½	—	—	—	—	B.f.	51	..	—	—	—	—	B.sf.
21	E.1 E.m.½	—	—	—	—	B.sf.	55	..	—	—	—	—	B.sf.

Case Miller, G., aged 24.—Patient, who had never been abroad before, left England on June 7, 1915, and went direct to the Peninsula, where he remained five months. While there he had a slight attack of dysentery, but had no emetin. He was invalided to Malta for anæmia and debility and thence was transferred to Alexandria in February, 1916. On May 19, during the routine examination of the Royal Army Medical Corps Staff at Mustapha Camp, patient was found to be a carrier of *E. histolytica*. He was kept under observation till May 30, when a 12-day course of emetin was commenced (one grain injection in the morning and ½ grain in keratin-coated tabloid each night). During the course patient was kept in bed on milk diet. He vomited on only one occasion. The *E. histolytica* infection disappeared after the third day of treatment and did not recur during a control of over one month, three weeks of which patient spent in the convalescent camp on light duty. The emetin had no effect on patient's temperature or pulse-rate.

Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool
		E.h.c.	E.f.				E.h.c.	E.f.	
1	..	—	—	B.sf.	22	E.1 E.m.½	—	—	L.b.uf.
2	..	+	—	B.sf.	23	E.1 E.m.½	—	—	B.uf.
4	..	+	—	B.sf.	24	E.1 E.m.½	—	—	B.uf.
5	..	++	—	B.sf.	25	..	—	—	B.sf.
6	..	++	—	B.sf.	26	..	—	—	B.sf.
7	..	++	—	B.sf.	28	..	—	—	B.sf.
8	..	++	—	B.sf.	29 C.	..	—	—	B.sf.
9	..	++	—	B.sf.	31	..	—	—	B.sf.
11	..	++	++	B.f.	33	..	—	—	B.sf.
12	..	++	—	B.uf.	35	..	—	—	B.sf.
13	E.1 E.m.½. V. 2hrs.	+++	—	B.uf.	37	..	—	—	B.sf.
14	E.1 E.m.½	+++	—	B.uf.	39	..	—	—	B.sf.
15	E.1 E.m.½	++	—	B.f.	42	..	—	—	B.sf.
16	E.1 E.m.½	—	—	B.f.	43	..	—	—	B.sf.
17	E.1 E.m.½	—	—	L.b.sf.	45	..	—	—	B.sf.
18	E.1 E.m.½	—	—	L.b.sf.	47	..	—	—	B.sf.
19	E.1 E.m.½	—	—	L.b.sf.	49	..	—	—	B.sf.
20	E.1 E.m.½	—	—	B.l.	51	..	—	—	B.sf.
21	E.1 E.m.½	—	—	B.sf.	55	..	—	—	B.sf.

Case Downs, J., aged 21.—Patient, who had never been abroad before, left England on April 2, 1916, and came direct to Egypt, where he arrived on April 12. On May 21, during the routine examination of the Royal Army Medical Corps Staff at Mustapha Camp, he was found to be a carrier of *E. histolytica*. He had not had dysentery but had had a severe attack of diarrhœa a fortnight after arrival in Egypt. The patient's stool was examined on three occasions during the four days preceding May 21, with negative results as regards *E. histolytica*. Patient was kept under observation till May 30, and was then given a 12-day course of emetin (one grain injection every morning and ½ grain in keratin-coated tabloid each evening). During treatment he was kept in bed on milk diet. There was no vomiting. The *E. histolytica* infection disappeared after the second day of treatment and did not recur during a control of over one month, three weeks of which patient spent in the convalescent camp on light duty. The emetin treatment had no effect on the patient's temperature or pulse-rate. An *E. coli* infection which was also present did not reappear after the treatment.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.h.c.	E.c.c.	E.f.				E.h.c.	E.c.c.	E.f.	
1	..	—	+	+++	B.uf.	23	E.1 E.m.½	—	—	—	L.b.uf.
3	..	—	++	—	B.uf.	24	E.1 E.m.½	—	—	—	L.b.f.
4	..	—	++	—	B.uf.	25	E.1 E.m.½	—	—	—	B.uf.
5	..	++	+	—	B.uf.	26	..	—	—	—	B.f.
6	..	+++	+	—	B.uf.	28	..	—	—	—	B.uf.
7	..	+++	—	—	B.uf.	30	..	—	—	—	B.uf.
8	..	+++	+	—	B.uf.	31	..	—	—	—	B.uf.
9	..	++	—	+++	B.uf.	32 C.	..	—	—	—	B.f.
10	..	—	—	+	B.uf.	33	..	—	—	—	B.sf.
12	..	—	—	—	B.uf.	35	..	—	—	—	B.sf.
13	..	+++	—	—	B.uf.	37	..	—	—	—	B.sf.
14	E.1 E.m.½. V. 1½ hrs.	+++	—	++	B.uf.	39	..	—	—	—	B.sf.
						41	..	—	—	—	B.sf.
15	E.1 E.m.½	++	—	—	B.uf.	43	..	—	—	—	B.sf.
16	E.1 E.m.½	—	—	—	B.uf.	45	..	—	—	—	B.sf.
17	E.1 E.m.½	—	—	—	B.uf.	47	..	—	—	—	B.sf.
18	E.1 E.m.½	—	—	—	B.uf.	49	..	—	—	—	B.sf.
19	E.1 E.m.½	—	—	—	B.uf.	51	..	—	—	—	B.sf.
20	E.1 E.m.½	—	—	—	B.uf.	53	..	—	—	—	B.sf.
21	E.1 E.m.½	—	—	—	L.b.sf.	55	..	—	—	—	B.sf.
22	E.1 E.m.½	—	—	—	L.b.uf.	57	..	—	—	—	B.sf.

Case Hollow, F., aged 23. Patient, who had never been abroad before, left England in June, 1915, and went direct to the Peninsula, where he had dysentery and was given 14 injections of emetin. He was invalided to Malta towards the end of December and there again developed dysentery, for which he was given a second course of 14 emetin injections. He came to Egypt in March, 1916, where, during the routine examination of the Royal Army Medical Corps Staff at Mustapha Camp, on May 16, he was found to be passing a stool containing blood and mucus and had a large infection of amœbæ, many of which contained red blood corpuscles. Trichomonas was also present. Patient was kept under observation till May 30, and was then given a 12-day course of emetin (one grain injection in the morning and ½ grain in keratin-coated tabloid by the mouth at night). During the treatment he was kept in bed on milk diet. The emetin caused no vomiting. The amœbæ disappeared after the second day of treatment and did not recur during a control of over one month, three weeks of which patient spent in the convalescent camp on light duty. The emetin course had no effect on the patient's temperature or pulse-rate. The case is of interest as no cysts of *E. histolytica* were

seen, though the stools were examined continuously for a fortnight before treatment was begun. Entamœbæ with included red-blood corpuscles were constantly present. The trichomonas infection was present before, during, and after treatment, but disappeared later.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.c.c.	E.f.	Trich.				E.c.c.	E.f.	Trich.	
1	..	—	+++ r.b.c.	+	B.uf. B.m.	22	E.1 E.m.½	—	—	+	B.uf.
						23	E.1 E.m.½	—	—	++	B.uf.
2	..	—	++	+	B.uf.	24	E.1 E.m.½	—	—	—	B.uf.
3	..	—	+	—	B.uf.	25	E.1 E.m.½	—	—	—	B.uf.
9	..	—	+++	++	B.uf.	26	E.1 E.m.½	—	—	—	L.b.uf.
10	..	—	+++ r.b.c.	—	B.uf.m.	27	..	—	—	—	B.uf.
						28	..	—	—	+++	B.uf.
11	..	—	+++ r.b.c.	—	B.uf.m.	29	..	—	—	+++	B.uf.
						30 C.	..	—	—	—	B.uf.
12	..	—	+++	—	B.uf.	32	..	—	—	—	B.sf.
13	..	—	+++ r.b.c.	—	B.uf. B.m.	34	..	—	—	—	B.sf.
						36	..	—	—	—	B.sf.
14	..	—	+++	—	B.uf. B.m.	38	..	—	—	—	B.sf.
15	E.1 E.m.½. V. 1 hr.	—	++	—	B.uf. B.m.	40	..	—	—	—	B.sf.
						44	..	—	—	—	B.sf.
16	E.1 E.m.½	—	+	—	B.uf.m.	46	..	—	—	—	B.sf.
17	E.1 E.m.½	—	—	+	B.uf.	52	..	—	—	—	B.sf.
18	E.1 E.m.½	—	—	+	B.uf.	54	..	—	—	—	B.sf.
19	E.1 E.m ½	—	—	++	B.uf.	56	..	+	—	—	B.sf.
20	E.1 E.m.½	—	—	++	L.b.l.	58	..	++	—	—	B.sf.
21	E.1 E.m.½	—	—	—	B.uf.						

Case Russell, H., aged 27.—Patient, who had not been abroad before, left England in June, 1915, and went direct to the Peninsula. In September he was invalided to Egypt for dysentery, for which he was given a course of emetin in Cairo. He was transferred to duty on the western Egyptian frontier, where he again had dysentery. He was sent back to Alexandria, where he was given another course of emetin. He was in the convalescent camp at Luxor and then was on duty at Mustapha (Alexandria), where he had a third attack of dysentery and was admitted to the Orwa-el-Waska Hospital on April 24, 1916. He was passing blood and mucus, in which active free amœbæ occurred in large numbers, many of which included red blood corpuscles. The next day no amœbæ could be found, but on April 26 amœbæ with included red blood corpuscles were again present in the mucus but not in the fæcal part of the stool. There were no cysts. Patient was given a 12-day course of emetin (one grain injection each morning and ½ grain by the mouth in tinc. opii mixture each evening); patient was kept in bed on milk diet. There was no vomiting. The amœbæ were not found after the second day of treatment, though some blood and mucus were found for some days after this. The stools eventually became normal and patient went to the convalescent camp, where cysts of *E. histolytica* appeared just a fortnight after the completion of the course of emetin. Patient continued to pass cysts and amœbæ till, on June 10, he was re-admitted to hospital with a recurrence of the dysentery. The stool contained blood and mucus and only free amœbæ in large numbers. Patient was then given a 12-day course of methyl emetin sulphate (one grain injection each morning and one grain in keratin-coated tabloid by the mouth each night). He was kept in bed on milk diet. There was no desire to vomit and no feeling of nausea, as after the emetin hydrochloride by the mouth. The amœbæ disappeared after the fourth day of treatment but cysts of *E. histolytica* and free forms were again present in large numbers three days after the course was finished. Neither course of emetin had any effect on patient's pulse-rate, but a slight irregularity in the temperature was noticed.

Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool
		E.h.c.	E.f.				E.h.c.	E.f.	
1	..	—	+++ r.b.c.	B.m.	22	..	—	—	L.b.uf.
2	..	—	—	B.uf. B.m.	23	..	—	—	B.uf.
3	..	—	++ r.b.c.	B.uf. B.m.	29	..	++	—	B.f.
4	E.1 E.m.½	—	+++	B.uf.	32	..	+	++	B.uf.
5	E.1 E.m.½	—	—	L.b.l.	34	..	++	—	B.f.
6	E.1 E.m.½	—	—	L.b.uf.	36	..	+	++	B.sf.
7	E.1 E.m.½	—	—	L.b.uf.	48	..	—	+++	B.uf. B.m.
8	E.1 E.m.½	—	—	L.b.uf.	49	M.E.1 M.E.m.1	—	+	B.uf.
9	E.1 E.m.½	—	—	L.b.uf.m.	50	M.E.1 M.E.m.1	—	—	—
10	E.1 E.m.½	—	—	B.f. B.m.	51	M.E.1 M.E.m.1	—	++	B.uf.
11	E.1 E.m.½	—	—	B.l.	52	M.E.1 M.E.m.1	—	++	B.uf.
12	E.1 E.m.½	—	—	—	53	M.E.1 M.E.m.1	—	—	B.uf.
13	E.1 E.m.½	—	—	B.uf.	54	M.E.1 M.E.m.1	—	—	B.uf.
14	E.1 E.m.½	—	—	L.b.uf.	55	M.E.1 M.E.m.1	—	—	B.uf.
15	E.1 E.m.½	—	—	—	56	M.E.1 M.E.m.1	—	—	B.uf.
16	..	—	—	L.b.uf.	57	M.E.1 M.E.m.1	—	—	B.uf.
17	..	—	—	B.l.	58	M.E.1 M.E.m.1	—	—	—
18	..	—	—	B.sf.	59	M.E.1 M.E.m.1	—	—	B.uf.
19	..	—	—	L.b.l.	60	M.E.1 M.E.m.1	—	—	B.uf.
20	..	—	—	B.uf.	63	..	++	+	B.uf.
21	..	—	—	B.uf.	65	..	+++	++	B.uf.

Case Greenwood, H., aged 19.—Patient, who had never been abroad before, left England in October, 1915, and went direct to the Peninsula. He remained there till December, when he was transferred to Mudros. In February, 1916, he came to Egypt. At Suez, in May, he had his first attack of dysentery, which lasted 18 days. He was not given emetin. He was sent to convalescent camp, where he again had dysentery and was admitted to hospital in Alexandria on June 23, where he was found to be passing blood and mucus stools containing free amœbæ with included red blood corpuscles. From June 26 onwards he was given a 12-day course of emetin (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth at night). There was no vomiting. The *E. histolytica* disappeared after the second day of treatment but reappeared before the course was completed, when cysts of *E. histolytica* were found for the first time. The infection again disappeared but recurred again a fortnight later. During the treatment the patient was kept in bed on milk diet. There was no change in the temperature or pulse-rate during the emetin course.

Days	Treatment	Findings		Stool	Days	Treatment	Findings		Stool
		E.h.c.	E.f.				E.h.c.	E.f.	
1	..	—	+++ r.b.c.	L.b.uf.B.m.	15	E.1 E.m.½	—	++	B.uf.
2	..	—	—	B.uf.B.m.	16	..	—	+++	B.uf.
3	..	—	++	B.uf.B.m.	17	..	—	—	B.uf.
4	E.1 E.m.½	—	+ r.b.c.	B.uf.B.m.	18	..	—	—	B.uf.
5	E.1 E.m.½	—	+	B.l.	19	..	—	—	B.uf.
6	E.1 E.m.½	—	—	B.uf.	20	..	—	—	B.uf.
7	E.1 E.m.½	—	—	B.uf.	21	..	—	—	B.uf.
8	E.1 E.m.½	—	—	—	28 C.	..	—	++	B.uf.
9	E.1 E.m.½	—	—	B.uf.	30	..	++	++	B.uf.
10	E.1 E.m.½	—	—	B.uf.	32	..	+++	—	B.uf.
11	E.1 E.m.½	—	—	B.uf.	34	..	+++	+	B.uf.
12	E.1 E.m.½	—	—	B.uf.	35	..	+++	++	B.uf.
13	E.1 E.m.½	—	++	B.uf.	36	..	+++	+	B.uf.
14	E.1 E.m.½	++	+++	B.uf.					

Case Jackson, H., aged 24.—Patient, who had previously served in India (five years) left England in September, 1915, and went direct to the Peninsula, where he remained till he was sent to Malta on account of dysentery, which persisted off and on till February, 1916. On

May 2 he came to Alexandria and was admitted to hospital for dysentery on May 17. Thence he went to convalescent home, and on July 5 was again admitted to hospital for the same disease. He was found to be passing stools with blood and mucus containing numerous active amœbæ, many of which had included red blood corpuscles. There was also a large lamblia infection. There had been no previous emetin treatment. On July 9 was commenced a 12-day course of emetin (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth at night). There was vomiting only on one occasion after the emetin. The patient was kept in bed on milk diet. The amœbæ disappeared after the second day of treatment while the lamblia infection persisted. This case was only controlled for one week after treatment, but it is of interest as an illustration of a case in which cysts of the amœbæ were never found. There was no relapse during the one week's control. The emetin did not affect the patient's temperature or pulse-rate.

Days	Treatment	Findings			Stool	Days	Treatment	Findings			Stool
		E.f.	L.c.	L.f.				E.f.	L.c.	L.f.	
1	..	+++ r.b.c.	+++	—	B.uf. B.m.	12	E.1 E.m.½	—	+++	—	B.uf.
2	..	+++ r.b.c.	+	—	B.uf. B.m.	13	E.1 E.m.½	—	+++	++	B.uf.
3	E.1 E.m.½. V. 2 hrs.	+++ r.b.c.	—	—	B.uf. B.m.	14	E.1 E.m.½	—	—	—	—
						15	..	—	+++	—	B.sf.
4	E.1 E.m.½	++ r.b.c.	—	—	B.uf.m.	16	..	—	++	—	B.uf.
5	E.1 E.m.½	—	—	—	B.l.	17	..	—	++	—	B.l.
6	E.1 E.m.½	—	—	—	B.uf.m.	18	..	—	+++	—	B.uf.
7	E.1 E.m.½	—	—	—	B.l.	19	..	—	+++	—	B.uf.
8	E.1 E.m.½	—	++	—	B.uf.	20	..	—	+++	—	B.uf.
9	E.1 E.m.½	—	+++	—	B.uf.	21	..	—	++	—	B.uf.
10	E.1 E.m.½	—	+++	—	B.uf.	22	..	—	+++	—	B.uf.
11	E.1 E.m.½	—	+++	—	B.uf.	23	..	—	+++	+	B.uf.

Case Wilkinson, E., aged 20.—Patient, who had not been abroad before, left England in May, 1916, and came direct to Egypt, where he remained till September, when he returned to England. He came back to Egypt again in January, 1916. Patient, who had never had dysentery before, was admitted to hospital with this disease on June 17, and was found to be passing stools containing blood and mucus and active amœbæ with included red blood corpuscles. He was found to have also an *E. coli* infection and later a tetramitus infection. He was given a 12-day course of emetin (one grain injection each morning and ½ grain in keratin-coated tabloid by the mouth at night). There was vomiting on only one occasion as a result of the treatment, during which patient was kept in bed on milk diet. As an *E. coli* infection was present and cysts of *E. histolytica* did not occur, it was difficult to judge the action of the treatment on the *E. histolytica* infection. However, cysts of *E. histolytica* were found for the first time three weeks after the course was completed. The emetin produced no alteration in the patient's temperature or pulse-rate. During the control of this case an interval occurred between the 23rd and 37th days owing to an accidental discharge from hospital.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	
1	..	—	—	+ r.b.c.	—	—	B.uf. B.m.
2	..	—	—	—	—	—	B.uf.
4	..	—	+	++	—	—	B.uf.
5	E.1 E.m.½	—	+++	+++	—	—	B.uf.
6	E.1 E.m.½	—	++	++	—	—	B.uf.
7	E.1 E.m.½	—	—	—	—	—	—
8	E.1 E.m.½. V. 2 hrs.	—	—	—	—	—	—
9	E.1 E.m.½	—	—	—	—	—	—
10	E.1 E.m.½	—	—	—	—	—	B.uf.
11	E.1 E.m.½	—	—	—	—	—	—
12	E.1 E.m.½	—	—	+	—	—	B.uf.

Days	Treatment	Findings					Stool
		E.h.c.	E.c.c.	E.f.	Tet.c.	Tet.f.	
13	E.1 E.m.½	—	—	—	—	—	—
14	E.1 E.m.½	—	—	++	—	—	B.uf.
15	E.1 E.m.½	—	—	++	—	+++	B.uf.
16	E.1 E.m.½	—	++	+++	—	+++	B.uf.
17	..	—	++	++	—	+++	B.uf.
20	..	—	—	—	—	+++	B.uf.
21	..	—	—	—	—	+++	B.uf.
23	..	—	+++	—	+++	—	B.uf.
24	..	—	+++	—	++	+++	B.uf.
37	..	—	+++	++	++	+++	B.uf.
38	..	—	+++	++	++	++	B.uf.
39	..	++	++	+++	+	+++	B.uf.

Section IV.

Case Hirst, aged 46.—Patient, who had previously served in India, South Africa and Egypt, left England in November, 1914, for France, where he remained till he was transferred to the Peninsula in August, 1915. He was there for nine weeks and was then in Mudros for two months, after which he came to Egypt, where, on May 10, during the routine examination of cooks in Mazarita Camp, he was found to be a carrier of *E. histolytica*. There was no history of dysentery. Patient was kept under observation in hospital, and here the *E. histolytica* cysts, which were of the small variety, disappeared from the stool and did not recur during one month's control. During the observation, the stool being examined every day, the patient suddenly developed an infection of *Waskia intestinalis*. It is curious that the only other case of infection which has been met with was then under observation in the same ward and occupying the bed next but one to this patient. It seems probable that the one case contracted the infection from the other while in hospital. The case was eventually discharged from hospital without treatment of the *E. histolytica* infection, which had not recurred. There were no symptoms attributable to it or to the Waskia infection, which was still present.

Days	Findings					Stool	Days	Findings					Stool
	E.h.c.	E.c.c.	E.f.	W.c.	W.f.			E.h.c.	E.c.c.	E.f.	W.c.	W.f.	
1	+++	+	—	—	—	B.uf.	20	—	—	—	—	—	B.uf.
5	++	—	+	—	—	B.uf.	21	—	+	—	—	—	B.uf.
6	++	—	—	—	—	B.uf.	22	—	—	—	+++	+++	B.uf.
7	+	—	++	—	—	B.l.	23	—	—	—	+++	+++	B.l.
9	+	—	+	—	—	B.l.	25	—	++	++	+++	+++	B.uf.
11	—	—	+	—	—	B.l.	26	—	—	—	+++	+	B.l.
12	—	—	—	—	—	B.l.	28	—	—	+	++	+	B.uf.
13	—	—	++	—	—	B.l.	29	—	—	—	+++	+	B.uf.
14	+	—	—	—	—	B.l.	30	—	—	+	—	—	B.uf.
15	—	—	+	—	—	B.uf.	31	—	—	—	++	++	B.uf.
16	—	—	—	—	—	B.l.	32	—	—	—	+	—	B.uf.
17	—	—	—	—	—	B.l.							

Section V.

Case Gildel, H., aged 20.—Patient, who had not been abroad before, left England in June, 1915, and spent four months at Anzac and Cape Hellas. He had diarrhœa with blood and mucus while there. He had no emetin. He came to Egypt towards the end of 1915, and during the routine examination of men in Camp A he was found to be passing a fluid stool in which were large numbers of tetramitus with lamblia and free amœbæ. The amœbæ turned out to be *E. coli*, and the three infections persisted in spite of attempts to get rid of them by means of emetin and β-naphthol. The case is of interest in showing the irregular occurrence of the flagellates in the stool. The patient was given a course of emetin by the mouth (½ grain a day for 12 days), and later a course of β-naphthol 15 grains t.d.s. for 12 days. The emetin had no effect on the temperature or pulse-rate, and did not produce vomiting. The patient was not kept in bed, and was given chicken diet.

Days	Treatment	Findings'						Stool
		E.c.c.	E.f.	L.c.	L.f.	Tet.c.	Tet.f.	
1	..	—	++	++	—	—	+++	Y.l.
5	..	++	—	++	—	+++	—	B.sf.
8	..	—	+	—	—	—	++	L.b.uf.
11	..	—	—	—	—	—	—	B.sf.
12	..	—	—	—	—	—	++	B.l.
13	..	—	—	—	—	—	—	B.uf.
14	..	—	—	—	—	—	+++	B.l.
15	..	—	+	—	—	—	—	B.l.
17	..	—	—	++	—	—	++	B.uf.
18	..	—	—	+++	+	—	+++	B.uf.
19	..	—	—	+++	—	—	++	B.uf.
22	..	—	—	++	—	—	+++	B.uf.
23	..	—	++	+++	+++	—	+++	B.l.
24	..	—	+	++	—	—	+++	B.l.
26	..	—	—	+	—	—	+++	B.uf.
28	..	—	—	+++	—	—	++	B.uf.
30	..	—	—	++	—	—	—	B.sf.
31	..	—	—	++	—	+	—	B.sf.
34	..	—	—	++	—	+	+	B.uf.
35	..	—	+	++	+	—	++	B.uf.
36	..	—	—	+++	—	—	—	B.sf.
37	..	—	—	++	—	—	+	B.uf.
38	E.m.½	+	+	+++	—	—	+	B.uf.
39	E.m.½	—	—	+++	—	—	—	B.sf.
40	E.m.½	—	—	+++	—	—	—	B.sf.
41	E.m.½	—	—	—	—	—	—	B.sf.
42	E.m.½	—	—	—	—	—	—	Y.uf.
43	E.m.½	—	—	—	—	—	—	L.b.uf.
44	E.m.½. V.10 min.	—	—	—	—	—	—	L.b.uf.
45	E.m.½	—	—	—	—	—	—	B.sf.
46	E.m.½	—	—	—	—	—	—	B.uf.
47	E.m.½	—	—	—	—	—	—	B.uf.
48	E.m.½	—	—	—	—	—	—	L.b.uf.
49	E.m.½	—	—	—	—	—	—	L.b.l.
50	E.m.½	—	+	++	—	—	—	L.b.uf.
51	E.m.½	—	+	+	—	—	—	L.b.l.
52	..	—	—	++	—	—	—	L.b.uf.
53	..	—	—	++	—	—	—	L.b.uf.
54	..	—	—	++	—	—	—	L.b.uf.
55	..	—	—	++	+	—	—	L.b.l.
56	..	—	—	—	—	—	—	B.uf.
57	..	—	—	—	—	—	—	L.b.l.
58	..	—	—	++	—	—	—	B.uf.
62	..	—	—	+++	—	—	—	B.sf.

Days	Treatment	Findings						Stool
		E.c.c.	E.f.	L.c.	L.f.	Tet.c.	Tet.f.	
63	..	—	—	+++	—	—	—	L.b.sf.
65	β-n.	—	—	+++	—	—	—	L.b.uf.
66	β-n.	—	—	—	—	—	—	L.b.uf.
67	β-n.	—	+	—	—	—	+++	L.b.uf.
68	β-n.	—	++	—	—	—	++	B.uf.
69	β-n.	—	—	—	—	—	—	—
70	β-n.	—	—	—	—	—	—	B.uf.
71	β-n.	—	—	++	—	—	—	L.b.uf.
72	β-n.	—	—	++	—	++	+++	Y.uf.
73	β-n.	—	—	++	—	++	+++	Y.uf.
74	β-n.	—	—	++	—	—	++	B.uf.
75	β-n.	+	—	+++	—	—	+++	B.uf.
76	..	—	++	+	—	—	+++	L.b.uf.
77	..	+	++	++	—	+	++	B.uf.
78	..	+	—	++	—	+	++	B.uf.
79	..	—	+	++	—	—	++	B.uf.
80	..	—	—	+++	—	—	++	B.uf.
81	..	—	—	+++	—	—	++	B.uf.
82	..	—	—	++	—	—	+	B.uf.
83	..	+	—	++	—	++	—	B.sf.
85 C.	..	+	—	—	—	—	+++	B.l.
88	..	+++	+	—	—	+++	+++	B.f.
90	..	+	+	—	—	+	+++	B.f.
91	..	+	+	+	—	+	+++	B.f.

Case Krechler.—Patient was admitted to hospital with diarrhœa. A large lamblia infection was discovered, and treatment with bismuth salicylate, 20 grains t.d.s., was instituted. The lamblia infection disappeared, but after ten days a *E. histolytica* infection became apparent.

Days	Treatment	Findings				Stool	Days	Treatment	Findings				Stool
		E.h.c.	E.f.	L.c.	L.f.				E.h.c.	E.f.	L.c.	L.f.	
1	B.s.	—	—	+++	—	B.l.	8	B.s.	—	++	+++	—	D.b.uf.
3	B.s.	—	—	+++	—	B.uf.	9	B.s.	—	+	+	—	D.b.uf.
4	B.s.	—	—	+++	—	B.uf.	10	B.s.	—	++	+	—	D.b.uf.
5	B.s.	—	—	+++	++	D.g.uf.	11	B.s.	—	+	—	—	D.b.uf.
6	B.s.	—	—	++	—	D.b.uf.	12	B.s.	++	+++	—	—	D.b.uf.
7	B.s.	—	++	++	—	D.b.uf.	13	B.s.	+	++	—	—	D.b.uf.

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